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SPECTRAL SENSITIVITY, AS MEASURED IN CEREBRAL VISUAL CENTRES

BY

DAVID H. INGVAR

LUND 1959

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SPECTRAL SENSITIVITY, AS MEASURED IN CEREBRAL VISUAL CENTRES

A Study of Responses to Monochromatic Flicker in the Unanaesthetized Cat

BY

DAVID H. INGVAR

LUND 1959
BERLINGSKA BOKTRYCKERIET

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Preface

The theme of the present investigation, a comparison between retinal and cerebral spectral sensitivity, was first taken up in 1954 in some preliminary experiments. In 1955 the main experimental series was carried out, and was concerned with a study of the responses to monochromatic light in the visual cortex, the lateral geniculate body and in the superior colliculus of the cat. Since the investigation was started several new findings on retinal mechanisms, as well as on cerebral responses to colour have appeared in the literature (p. 21—22), and they have been of great help in the analysis of the present results. During the course of the investigation preliminary reports of the findings have been given (INGVAR, 1956 a and b, 1957, 1958).

The requirements of the present investigation, including the advanced technical equipment necessary, were amply provided at the Nobel Institute for Neurophysiology in Stockholm, where there has long been such a deep knowledge of visual physiology. The author is very indebted to the Director of this Institute, Professor Ragnar Granit, for his inspiring interest and critical advice. Dr. G. B. Arden and Dr. B Donovan have been kind enough to read the manuscript and to offer much helpful discussion and criticism. Thanks are also due to Miss Gun Larsson and Miss Anne-Marie Bengtsson who gave technical assistance during the experiments and the histological work. The experimental records were mainly analyzed during author's stay at the Institute of Physiology of the University of Lund, and its head, Professor Georg Kahlson, is thanked for providing accommodation for the author. Miss Karin Hellmer gave secreterial assistance and Miss Birgit Sörensson drew several diagrams. Mr

Olle Jönsson helped with the photographic work. The costs of publication have been defrayed by the Swedish Medical Research Council which has also provided financial support for the work.

Lund, December 1958.

David H. Ingvar

Chapter I

Introduction

In species whose eyes contain rods and cones, measurements of visibility and of retinal sensitivity under conditions of dark adaptation usually show a spectral distribution which, provided appropriate corrections are made, is in good agreement with the absorption curve of rhodopsin (visual purple), the photopigment of the rods (KÖNIG 1894; *cf.* GRANIT 1955 a, DARTNALL 1957 and others). This fact has generally been held to show that in the dark adapted state, when vision is achromatic, light reception is determined by one photopigment only, namely rhodopsin.

However, electrophysiological findings in several species with rods and cones have demonstrated that in the scotopic state (defined as the one in which the "photopic dominator" (GRANIT 1945) is not obtainable) responses from single retinal "elements" (probably larger ganglion cells), or even the ERG response, may not always express a spectral sensitivity identical to the rhodopsin curve (GRANIT 1955 a; *v. i.*, p. 15—17). These results thus indicate that the receptor mechanism underlying scotopic vision is complex, and that some, at least, of its components depend upon interference, chemical or neural, of photopigments with absorption characteristics different from those of rhodopsin *in vitro*.

Since scotopic vision in man and animals nevertheless so closely mimics the rhodopsin absorption curve, it might be argued that retinal information of non-rhodopsin character is lost during transmission of optical impulses to the brain or disregarded in the process of perception. The discrepancy between some of the retinal findings and the evidence from visibility measurements can only be settled by an investigation

under conditions of dark adaptation of the transmission of the visible spectrum to the visual centres of the brain.

In the present work light reception has been studied in the dark adapted cat by means of potentials evoked in visual centres of the brain, using the ERG as a peripheral control. Diffuse illumination of the retina by intermittent monochromatic light of known intensity was used. Spectral sensitivity was calculated from relative quantum intensity reciprocals to cerebral responses equal in amplitude, or latency, in the wavelengths tested.

If the optical messages under conditions of dark adaptation depend upon a homogeneous receptor mechanism, then all visually evoked responses in the brain to diffuse retinal illumination must of necessity indicate one form of spectral sensitivity, no matter what index is measured (*e. g.* amplitude or latency of On or Off components). The cerebral sensitivity distribution should also, at least grossly, show agreement with the spectral sensitivity curve expressed by the ERG in a given experiment.

However, it will be demonstrated below that in the dark adapted state the spectral patterns found in the brain of the unanaesthetized cat with low intensities of flicker only occasionally, and partially, correspond to the curve of retinal sensitivity in a given experiment. With the method used, it was found that retinal, and particularly cerebral spectral curves showed deviations from the rhodopsin absorption spectrum. In the visual centres of the brain, however, some of these deviations showed a systematic change with increasing intensity of flicker, *i. e.* with increasing light adaptation. The findings suggest the existence of at least two spectral sensitivity patterns at the lower intensities of flicker, *i. e.* at lower degrees of light adaptation. These patterns have been termed scotopic. Evidence for an interaction between the receptor mechanisms represented by these two low-intensity patterns will also be presented.

At higher intensities of flicker, a third pattern was found which was accentuated by modest additional adaptation to white light. This pattern was defined as the photopic one since it contained a peak at 550 mμ, which is the spectral location of the "photopic dominator" peak of GRANIT (1945).

In general, the findings are consistent with the idea of a complex nature of the scotopic light reception mechanism. They also seem to add new information on retinal events during the transition from scotopic to photopic conditons. Since responses from all the main visual centres were analyzed, it will also be possible to reach some general conclusions regarding the neural transmission of the visible spectrum to the brain in the cat.

A. THE CAT'S VISUAL SYSTEM

In this section some relevant information concerning the anatomy and physiology of the visual system of the cat has been summarized briefly. This summary will also form the basis for the discussion of the results in Chapter IV.

1. *Anatomy*

For a complete survey of the visual system of the carnivores and other vertebrates, reference is made to "The Vertebrate Eye" by G. L. WALLS (1932) and to the recent monumental work "The Vertebrate Visual System" by the late STEPHEN POLYAK (1957) in which this subject is treated in its complete biological context.

The retina. Earlier workers have described both rods and cones in the cat's retina and studied their number and distribution. SCHULTZE (1866) reported the ratio of rods to cones to be about 2 to 3 times higher than that of man. There is evidence that the area centralis of the cat has a higher concentration of cones than the periphery (ABELSDORFF 1905, KOLMER 1936). In general, the cat's retina resembles the periphery of that of the human (*cf.* GRANIT 1943, WILLMER 1955).

In view of the results to be presented below, it is of interest that two kinds of rods, pink and green, have been found in the frog (BOLL 1876, KÜHNE 1878, DENTON and WYLLIE 1955). The pure rod retina of the guinea pig also contains two distinct forms of rods which have been termed alpha- and beta-rods by SJÖSTRAND (1953) in his studies employing electron microscopy (*v. i.*, p. 89). It is not known, however, whether the rods in the cat can be differentiated in a similar way.

The organization of retinal receptors and neurons has a special significance for the present investigation in which visual responses were picked up with small surface electrodes, primarily from the

enlarged retinal projection area of the cerebral cortex. Such responses probably reflect activity in a limited retinal receptor population. Knowledge of the morphological relationships of the retinal elements in the cat is therefore of basic importance for the interpretation of the results obtained. However, no detailed study of retinal neuronal organization in the cat seems to exist. One has therefore to fall back upon analogies with other vertebrates (especially anthropoids and man) more closely investigated (WALLS 1932, POLYAK 1941, 1957). Some of the principles of retinal functional organization merit emphasis here.

The *rod receptor-conductor system* includes a number of bipolar ganglion cell types, most of which are of a diffuse type interconnected by dendritic expansions. These neurons synapse with diffuse ganglion cells which function as "huge collectors or summators of rod current of excitation" (POLYAK 1957). It is of interest that the rod system apparently includes at least two different types of pathways of which one, with a higher threshold, is less diffusely organized and probably involves the "mop" bipolars (HECHT *et al.* 1938, PIRENNE 1953, WILLMER 1955). GRANIT (1942) in cats and guinea pigs distinguished real rods from conelike rods. There is also evidence from cases of achromatopsia in man of two different rod mechanisms (HECHT *et al.* 1948, SLOAN 1954, YONEMURA and ISHISAKA 1955, *v. i.*, p. 90). RONCHI (1957) on the basis of ERG studies has recently distinguished two types of rods in man.

The *cone system,* on the other hand, in man and anthropoids partly utilises private "disynaptic" pathways to the optic nerves, as well as conduction through the same bipolars as those connected to rods. Even at the retinal level, there is anatomical evidence that in this convergence the cone system may influence the rod system while the opposite cannot take place (POLYAK 1957). But, as POLYAK (*l. c.*, p. 585) writes, "it is possible that under certain conditions of stimulation the entire receptor-conductor mechanism functions as a unit, in which the same diffuse bipolars serve as a common channel for both rod and cone influences (as it could, for example, during the transitional period from photopic to scotopic vision and vice versa)". Such a transitional state is of a special interest in the present study in which cerebral spectral sensitivity was investigated as a function of increasing intensity of flicker, *i. e.* during increasing light adaptation.

The relatively small number of cones in the cat's retina, in which there is no fovea, but an area centralis (CHIEVITZ 1889), as well as the whole organization of its central visual system (*v. i.*), makes it unlikely that this animal is equipped with a large number of private cone pathways of the "point to point" type via "midget" bipolars. Thus, one would not expect to find areas of pure cone function in central visual structures of the cat. Such areas seem to exist in, *e. g.*,

the monkey where the cortical macular fields are so enormous (ADRIAN 1946).

Finally, it should be mentioned that the retina possesses several types of neurons (amacrine and horizontal cells, "shrub" ganglion cells) which probably act as integrators distributing excitation and inhibition.

The optic nerve and tract. According to BRUESCH and AREY (1942) and to P. O. BISHOP *et al.* (1953 a) there are about 120,000 nerve fibres in the cat's optic nerve, a figure corresponding well to the number of ganglion cells, but contrasting with the much greater number of retinal receptors and of principal cells in the dorsal nucleus of the lateral geniculate body (LGN). According to P. O. BISHOP *et al.* (1953 a) the optic nerve fibres, which are all myelinated, can be separated into two main groups, one, with larger fibres (3—8.5 microns, mean conduction velocity 30—40 m/sec, maximum 70 m/sec.) and another with smaller fibres (1—2 microns, mean conduction velocity 15—20 m/sec.). Eighty per cent of all the fibres are below 3 microns in diameter. The thinner fibres are situated dorsally in the nerve (P. O. BISHOP 1953). It should be recalled that CHANG (1951, 1952, 1956) and CHANG and KAADA (1950) have produced evidence for a third, thicker, group of fibres in the optic nerve of the cat. These authors suggested that such a triple pathway might serve trichromatic colour vision, a theory which, however, has been criticized by several authors (*cf.* summaries by WALLS 1953 and ALBE-FESSARD 1957).

Recently G. H. BISHOP and CLARE (1955) have indicated the presence of 4 groups of fibres in the cat's optic tract. The fibres with largest diameter (conduction velocity 40—50 m/sec.) relay in layers A and A_1 of the LGN and go to the cortex. The next group (15—25 m/sec.) relays in layer B of the LGN and goes to the lateral nucleus of the thalamus whilst the two thinner groups go to the pretectal and tectal regions respectively. Nevertheless, it seems advisable to conclude that the optic nerve contains mainly two groups of fibres, thicker and thinner, the first being relayed mainly to the cortex but with branches to the colliculus (*cf.* GRANIT 1955 b), the second group containing the majority of the fibres and chiefly going to the brain stem.

The lateral geniculate body. The main part of the present investigation has been devoted to the retino-cortical pathway which synapses in the dorsal nucleus of the lateral geniculate body (LGN). Few parts of the central nervous system of the cat have been studied anatomically as carefully as the LGN (TELLO 1904, RAMON Y CAJAL 1952—1955, MINKOWSKI 1913, THUMA 1928, BARRIS 1935, O'LEARY 1940, GLEES 1941, P. O. BISHOP 1953). It consists of 3 definite "magnocellular" layers, and so differs from the LGN of primates and man in which there are 6 or more layers, of which usually only two are "magnocellular". Topographical studies in the monkey (CLARK and

PENMAN 1934) demonstrate that the foveal parts of the retina synapse in the middle of each layer, *i. e.* on the "top" of the LGN when exposed from above. A similar arrangement is present in the cat (MINKOWSKI 1920).

It seems proved that the principal cells of the LGN, which all project to the cortex, receive a main branch from the optic nerve fibres, usually of large diameter, while a thinner branch continues to the pretectal region and the superior colliculus in the same bundle as the direct (usually thinner) retinotectal fibres (O'LEARY 1940, BARRIS, INGRAM and RANSON 1935, G. H. BISHOP and CLARE 1955). In the cat, every optic fibre makes synaptic contact with about 10 different cell bodies in the same layer of the LGN (GLEES 1941), in an arrangement suggesting "partially shifting overlapping" (*cf.* O'LEARY 1940). All three layers contain numerous small neurons with intralaminar connections which, according to P. O. BISHOP (1953; *cf.* CHANG 1951), are responsible for repetitive firing in the postsynaptic neurons. The "point to point" connections, consisting of a single presynaptic terminal on only a few LGN cells, which exist in the monkey (GLEES and CLARK 1941) have not been observed in the LGN of the cat.

On the whole, the neuronal organization of the LGN of the cat, with its few "magnocellular" layers, and with the rather extensive overlap in the synaptic contacts between optic fibres and the LGN cells, might be taken as an expression of the domination of rod function in the visual system of this nocturnal animal. Similarities between the cat's LGN and that of rod-dominated nocturnal primates (Lemur, Perodicticus; HENSCHEN 1925, 1930, SOLNITSKY and HARMAN 1946) are, in fact, many and striking (*v. i.*, p. 13).

The pretectal area and the superior colliculus. The optic inflow to the brain stem may be regarded as the "paleovisual" system in contrast to the geniculo-cortical which corresponds to the "neo-visual" system (MARQUIS 1934). BARRIS, INGRAM and RANSON (1935), MAGOUN and RANSON (1935), BUCHER and BÜRGI (1950) and G. H. BISHOP and CLARE (1955) among others, have made detailed studies of the optic connections in the pretectum and the tectum. Visual fibres of the thicker and the thinner type (P. O. BISHOP 1953) terminate in both structures and they are topographically organized in the colliculus (APTER 1945). These fibres, together with cortico-pretectal and cortico-tectal connections, are, in addition, responsible for the innervation of "nonspecific" (reticular) visual pathways in medial parts of the higher brain stem (MAGOUN 1952, DELL 1952, INGVAR and HUNTER 1955). For reasons to be discussed below (p. 19), the "nonspecific" visual pathways could not be studied with the technique used in the present experiments.

The visual cortex. The cortical projection fields of the optic radiation fibres (area 17 of BRODMANN) in the cat have long been known

to be situated in the gyrus lateralis (CAMPBELL 1905, BRODMANN 1909). The cat's striate area is known in its detailed anatomy only to a limited extent, and anatomical (MINKOWSKI 1913) and physiological (MARSHALL and TALBOT 1941) workers have arrived at contradictory results regarding the topography of the retinal cortical projection fields. There is, however, on comparative anatomical grounds (POLYAK 1957) reason to presume that the projection field of the area centralis comprises a major part of the anterior striate area on the dorsal and mesial side of the hemisphere. RAMON Y CAJAL (1923) has given a very thorough description of the histology of the cat's visual cortex. He suggested that the stria of GENNARI — VICQ D'AZYR represented the layer in which the afferent optic fibres terminated. This was later proved to be correct (also in the cat) by POLJAK (1927) with the Marchi technique. He also demonstrated the strict topographical organization of the geniculo-cortical bundles, as well as the existence of some optical terminations in peri- and parastriatal areas (BRODMANN's fields 18 and 19).

The histology of the striate area in the cat shows several important differences from the picture met with in anthropoids and man. This is especially true for the 4th lamina, the "inner granular layer" (BRODMANN 1909), in which the optical terminations are found. This layer in the cat is comparatively simple and poor in smaller cells (RAMON Y CAJAL 1923, O'LEARY 1941).

The primary cortical visual projection area of the cat, like its LGN, has several characteristics in common with the nocturnal apes in which a "magnocellular" fourth layer is found also. From the investigations of HENSCHEN (1925, 1930, 1931) in nocturnal and diurnal primates it is clear that within the striate area a definite functional organization can be found which is strictly related to the anatomy of the retina. He demonstrated, in fact, the existence of an anatomical "duplicity" in the visual centres of the brain which could be related to the histology of the retina. Thus, nocturnal primates with rods, but without cones and macula, have a "magnocellular" inner granular layer consisting of large, so called *"Lichtzellen"*, with the larger cell bodies toward what corresponds to the periphery of the visual projection field. The diurnal forms (and man), on the other hand, have rods and cones and a macula. The 4th layer of their striate area contains both the large *"Lichtzellen"*, as well as numerous smaller densely stained cells, the so called *"Farbenzellen"* which are concentrated in the cortical macular field. These studies have been amply confirmed and extended by the detailed investigation of SOLNITZKY and HARMAN (1946; *cf.* WALLS 1953). Concerning the cat, there is evidence from RAMON Y CAJAL (1923) and also from O'LEARY (1941) that this animal (which is equipped with both rods and cones — with the cones being concentrated in and around the area centralis (ABELSDORFF 1905))

has a striatal organization according to the principle of HENSCHEN of cerebral visual duplicity. The projection field for areal vision (in and around what may correspond to the calcarine fissure) seems more "parvocellular" in character in its fourth layer, while the periphery contains larger cell bodies.

SUMMARY: *The cat's retina is dominated by rods although cones are also present. The cerebral visual system of the cat shows the characteristics of an animal of nocturnal habits with overlapping synaptic contacts in "magnocellular" geniculate and cortical relay stations, similar to that found in, e. g., lower nocturnal primates, which are also rod-dominated. Some anatomical evidence, however, indicates that "duplicity" is, to some extent at least, present also in cerebral parts of the cat's visual system. Thus, there are two main groups of fibers, thicker and thinner, in the optic nerve and tract, and, in the striate area, there is suggestive evidence of a "parvocellular" fourth layer in the central (areal) projection field and a "magnocellular" in the peripheral visual area.*

2. *Physiology*

In this section, some important facts from investigations in the cat concerning the light absorbing characteristics of the preretinal media, the spectral sensitivity of retinal elements, and the electrophysiolgy of the central visual pathways will be summarized. An extensive review of feline retinal mechanisms is to be found in the monographs by GRANIT (1947, 1955 a).

For the present purpose, cerebral visual activity was best studied with evoked responses to *intermittent* light stimulation, since maintenance of constant illumination does not give rise to changes of either cortical macrorhythms (CLAES 1939), or the discharge rate of individual cortical units (JUNG *et al.* 1952, HUBEL 1957). A brief recapitulation of current interpretations of evoked potentials in central visual pathways has therefore been added (*cf.* ALBE-FESSARD 1957) and the following section thus forms a basis for the discussion of the results which are based upon measurements of latency and amplitude of cerebral visual responses evoked by flicker.

The preretinal media and the tapetum. The preretinal media in the cat — especially the crystalline lens — have been studied by WEALE (1954) and recently also by DODT and WALTHER (1958 a). Like ROGGENBAU and WETTHAUER (1927) and LUDVIGH and MCCARTHY

(1938), in cattle and man respectively, these authors found an increased absorption of the cat's lens in the ultraviolet. The absorption in the cat was, however, for wave-lengths longer than 430 mμ less than 0.1 expressed in density and the spectral variation in density negligible. These facts explain why DODT and WALTHER (1958 a) did not find any differences in retinal spectral sensitivity between the cat's intact and the lensless eye, if moderate intensities were used. In the present experiments on cats with intact eyes, comparatively low intensities were used and no correction has therefore been made for the lens absorption. The calculations of relative intensity values have thus been made at the corneal level (*v. i.*, p. 31 and p. 80).

Another characteristic of the cat's eye to be considered is the tapetum. The reflective properties of this tissue have been studied *in situ* by WEALE (1953 a). He did not find tapetal reflection to have any effects upon spectral sensitivity curves under scotopic or photopic conditions, although a marked effect was found upon the absolute threshold. The tapetum has also recently been studied by DODT and WALTHER (1958 b) who investigated the photopic dominator of the cat by means of the ERG. They found that between 0.45 and 0.75 log units more light was necessary to evoke ERG responses of the same size from retinal areas outside the tapetum as compared to those within the tapetum itself. There were, however, no differences in spectral sensitivity in intratapetal and extratapetal areas. In the present investigation, cats with yellow and green tapeta were used. In view of the reports summarized above, however, it is believed that tapetal reflection has been of little importance for the results since mainly *relative* changes in responses from one and the same cerebral point during monochromatic flicker of increasing intensity were studied. Therefore, no corrections for this factor have been made in the calculations (see Discussion, p. 80).

The retina. Recently GRÜSSER (1957) in the cat succeeded in recording unit activity from a layer in the cat's retina which probably corresponds to that of horizontal and bipolar ganglion cells (MACNICHOL, MACPHERSON and SVAETICHIN 1957). His findings in many respects confirm in the cat the observations of SVAETICHIN (1956 a, b, c) who first demonstrated in fishes the longlasting depolarization in such units on illumination. It is not yet known, however, whether the cat's retina also possesses nervous elements, coupled to cones, in which stimulation with complementary colours produces potential phenomena of opposite signs (SVAETICHIN 1956 a, b; *cf.* MOTOKAWA *et al.* 1957).

In a large series of investigations in the cat and other species, GRANIT and his collaborators have determined spectral sensitivity distributions from the ERG, as well as from isolated larger retinal

ganglion cells ("retinal elements"; *cf.* GRANIT 1947, 1955 a and RUSHTON 1949, 1953 a). Spectral sensitivity is defined as intensity reciprocals (1/I) to *equal* responses (size of b-wave in ERG, spike frequency, etc.) at different wave lengths (GRANIT 1942, 1943, 1945, 1947, 1948, 1955 a, GRANIT and WIRTH 1953, DODT and WALTHER 1958 a, 1958 b, DONNER 1950, 1957, DONNER and GRANIT 1949, WIRTH 1953).

It is evident from this work that under scotopic conditions the retinal spectral sensitivity of the cat, measured with "single" flashes at threshold, usually shows a fairly good correspondence to the absorption curve of rhodopsin (visual purple, VP) or pigment 497 of DARTNALL (1953, 1957) which is probably identical to the visual purple of the cat (GRANIT 1955 a). The receptor mechanism underlying this broad curve has been called the *scotopic dominator* (GRANIT 1947). It should be noted, however, that the dominator was defined on the basis of work with single units.

It is of interest that spectral sensitivity determinations in the cat seemed to correspond best to the VP curve when "averaging" responses such as ERG responses to "single" flashes (WIRTH 1953, DODT and WALTHER 1958 b) were studied in the dark adapted state, or when the curve was calculated from several determinations on single units (DONNER and GRANIT 1949). Deviations from the VP curve were, in fact, recorded *under scotopic conditions,* especially in isolated Off and On/Off elements by DONNER and GRANIT (1949) in the cat (*cf.* also findings by DODT and ELENIUS (1956) and by DODT (1956 a) in the rabbit). The scotopic "blue-shift" of retinal sensitivity obtained from ERG responses to flicker by GRANIT and WIRTH (1953) in the cat might further be recalled to indicate that the scotopic receptor mechanism is a heterogenous one. This suggestion is substantiated by the results presented below.

In light adaptation in the cat, about 36 % of the retinal elements were found by GRANIT (1943) to demonstrate a broad sensitivity curve with its maximum at about 550—560 mμ. This curve represents the *photopic dominator* of the cat (GRANIT 1945) which was considered responsible for the Purkinje shift of spectral sensitivity. Recently DODT and WALTHER (1958 b) have studied this phenomenon in the cat by means of the ERG (*cf.* BARLOW, FITZHUGH and KUFFLER 1957).

More narrow sensitivity curves found by GRANIT and his coworkers (*cf.* DONNER 1957) under conditions of weak white, or selective monochromatic light adaptation, were interpreted as *modulator curves,* representing light receiving mechanisms with restricted sensitivity within the spectrum. "Pure" modulator curves with only one peak could easily be obtained in animals with a large number of cones. In the cat, however, indirect methods such as selective adaptation or polarization were found to be necessary to bring out modu-

lator curves. By direct recording they often occurred as narrow-banded humps on broad dominator curves. In various animals the modulator maxima were found in three preferential regions (440—460 mμ, 520—540 mμ and 580—600 mμ), and it was therefore assumed that the modulators might serve trichromatic colour vision (GRANIT 1945, 1947, 1955 a) while the broader dominator curves suggested their relation to brightness discrimination. The dominator-modulator theory and the photopigments will be discussed in relation to the findings of the present work in Chapter IV (p. 87).

Retinal mechanisms sensitive to specific wave-lengths in the cat have further been demonstrated by MOTOKAWA, IWAMA and EBE (1952). These workers found differences in time course to different colours when they measured the electrical excitability of the retina after a brief illumination with spectral light.

The behaviour of single retinal elements in the cat in flicker have been investigated by ENROTH (1952), KUFFLER (1953), DODT and ENROTH (1954) and by GRÜSSER and his associates (1957, 1958 a, b). It is clear that a difference of rod and cone fusion frequency can be demonstrated in plots of fusion frequency against light intensity. At the highest frequencies (cone flicker) On as well as Off effects were found to take part in the ERG-waves recorded. In single retinal elements in the dark adapted rabbit DODT (1956 a) further demonstrated the existence of inhibition of retinal spike activity with increasing intensity. Calculating two curves for spectral sensitivity, at the "excitatory" and the "inhibitory" flicker fusion points respectively for different wave-lengths, he found the two curves to be different. This, he concluded, showed a shift of emphasis from one type of photoreceptor to another when the flicker intensity was raised. The spectral curves differed, however, from both the photopic and the scotopic curves obtained from threshold measurements. The sensitivity curve at "inhibitory fusion" (highest intensity) showed marked peaks in the blue (450—470 mμ) and the green (520 mμ). High sensitivity was sometimes also found in the violet. The findings of DODT (1956 a) emphasize the inhibitory interaction between rods and cones (ADRIAN and MATTHEWS 1928, GRANIT and RIDDELL 1934, GRANIT 1947) for which, as mentioned, there is also some anatomical evidence (POLYAK 1957).

A note might here be added on the exogenous "centrifugal" fibers to the retina (RAMON Y CAJAL 1952—1955, GRANIT 1954, 1955 b, DODT 1956 b, HERNÁNDEZ-PEON *et al.* 1957). It is believed that such fibres, present also in other afferent systems, may exert a control upon the receptor and the centripetal messages. Several investigations have established the correlation between such a centrifugal control and the general state of central nervous excitability, as measured by the EEG (*cf.* review by FRENCH 1957). In the present experiments

the centrifugal influences upon the retina have probably been of little importance due to the fact that stable cerebral excitatory conditions were attained by means of the *cerveau isolé*-technique of BREMER (1935).

Conduction in the optic nerve and tract. Signals leaving the retina reach the optic nerve after shorter or longer latency depending upon the intraretinal conduction time which may vary considerably (ADRIAN 1941, DONNER 1950, DODT 1956 b). HARTLINE (1941) in his classical investigations, early established the relationship between intensity of illumination and frequency of discharge in the optic nerve fibres and he also studied the dependence of latency upon intensity. ADRIAN (1946) pointed out the difficulties in proving a rod and cone component in the response to a flash picked up from the whole optic nerve in the cat (*cf.* DODT and ENROTH 1954). In single units, however, DONNER (1950) demonstrated in the cat differences in signal pattern for different wave-lengths. He found that On/Off units reached a maximum in their spike frequency-time curves, faster for red light than for green and for blue. He expressed the view that brightness is communicated by absolute frequency of discharge while colour is transmitted by frequency modulation. Related findings have recently been reported by LENNOX (1958 a and b, *cf.* p. 21).

Corresponding to the fibre groups in the optic nerve and tract (*v. s.*) a typical potential pattern can be recorded in these structures on electrical stimulation (O'LEARY and BISHOP 1939, P. O. BISHOP 1953, G. H. BISHOP and CLARE 1955, CHANG 1956, LENNOX 1958 a and b). The relationship between the fibre groups of the optic nerve and the rod-cone mechanisms of the cat's eye is unknown at present.

The lateral geniculate body. Several studies, especially by G. H. BISHOP and O'LEARY (1940, 1942 a and b; *cf.* G. H. BISHOP and CLARE 1955) and by P. O. BISHOP (1953) and his coworkers (1953 a and b, 1954, 1956) both with gross surface leads, and with penetrating micro-electrodes, have been devoted to the pre- and postsynaptic potential of the LGN. In some experiments of the present study potential variations were recorded by a gross lead placed on the surface of the LGN exposed from above by cortical resection. The potentials recorded were of the same type as those depicted by LENNOX (1956) consisting mainly of a negative phase followed by a more or less multiphasic positive envelope. Latency to the first peak as well as over all peak to peak (positive to negative peak) amplitude were measured. From the analysis of P. O. BISHOP (1953) it could be argued that changes in latency of the positive peak express changes in presynaptic events in large optic tract fibres, while changes in amplitude might concern postsynaptic (optic radiation) events in addition. Differences between the two might therefore express changes in spectral sensitivity taking place at the synapse, *i. e.* it would be possible to find out by differential measurements of charac-

teristics of the surface LGN response whether changes ("amplification") of spectral humps are taking place during the passage through the LGN. Uncertainty as to electrode placement, factors of injury, threshold to illumination in fibres of different conduction velocity etc., make this hypothesis adventurous, however, when supported only by data from gross surface recordings.

The superior colliculus. A small lead placed on the surface of superior colliculus picks up a composite response on light stimulation (G. H. BISHOP and O'LEARY 1938, 1940, 1942 a and b) in which early components might be due to corticofugal feed-back through striatotectal connections (INGVAR and HUNTER 1955). Collicular responses due to fast and slow retinotectal fibres (P. O. BISHOP 1953, *cf.* G. H. BISHOP and CLARE 1955) were therefore studied in acute and chronic preparations after ablation of the visual cortex bilaterally (*cf.* HUNTER and INGVAR 1955). Uncertainty as to electrode placement after such ablations did not make it possible to relate the results to the topographical findings of APTER (1945; *cf.* HAMDI and WHITTERIDGE 1954).

Intense light stimulation gives rise to very well defined visual responses deep in the medial brain stem in unanaesthetized preparations where there is an "unspecific" visual (reticular) pathway (MAGOUN 1952, DELL 1952, INGVAR and HUNTER 1955). This interesting pathway did not yield responses suitable for measurement with the technique chosen (stereotaxically oriented steel needles). Probably a very intense light source, flashing at a low frequency, and a special recording technique is necessary for an investigation of spectral sensitivity in these unspecific visual pathways which, by their presumed relation to conscious perception, may well be of some interest for the colour vision problem.

The visual cortex. Numerous older and more recent studies have dealt with the functional significance of the various components of the response in the visual area as evoked by electrical stimulation of the optic tract, nerve, or radiation and recorded by a surface or a penetrating electrode. It seems proved that the first two positive deflections of the response constitute arrival volleys of different velocity to the 4th layer of the striate area (CHANG and KAADA 1950, MALIS and KRUGER 1956, BREMER and STOUPEL 1956). There is, furthermore, evidence for a postsynaptic intracortical origin of components 3 and 4 (surface positive) as well as for component 5 (surface negative) (CLARE and G. H. BISHOP 1952, G. H. BISHOP and CLARE 1952).

Light stimulation, on the other hand, may give rise to visual cortical responses of very variable form in which the 5 components are not always present (ADRIAN 1941, MARSHALL, TALBOT and ADES 1943), or vary with intensity and wave-length (CHANG 1952, LENNOX and MADSEN 1955, MADSEN and LENNOX 1955; *cf.* p. 21). Repetitive

responses may also occur which probably are related to repetitive firing in the LGN (P. O. BISHOP 1953) as well as to an interaction with intrinsic rhythmic phenomena in the cortex (BARTLEY 1936, CLARE and BISHOP 1952, 1955). Present knowledge of the 5 components of the striatal response cannot therefore be immediately applied to the many variations of cortical responses to flicker which were recorded by surface electrodes in the present work. As it will appear, no definite conclusions can be reached as to pre- or postsynaptical location of certain cortical spectral sensitivity patterns based upon gross measurements of the latency or amplitude of potentials evoked by different wave-lengths (*cf.* LGN-responses, p. 18). It may only be assumed that the responses studied were, in part at least, related to postsynaptic activity of cortical On, Off, and On/Off units of the types first described by JUNG, v. BAUMGARTEN and BAUMGARTNER (1952) and studied by HUBEL (1957), CREUTZFELDT and AKIMOTO (1958) and by GRÜSSER and CREUTZFELDT (1957).

The present study concerns only visual area I (MARSHALL, TALBOT and ADES 1943) and mostly recordings from the anterior parts. With the light source used, visual area II did not yield responses large enough to be studied systematically. The anterior part of visual area I probably represents the projection field of the lower nasal quadrant of the contralateral retina (MARSHALL and TALBOT 1941, *cf.* contradictory evidence in MINKOWSKI 1913) which is equipped with a tapetum. Responses in this cortical area were of large size, probably in part due to tapetal reflection.

Finally, it should be recalled that different points in the visual cortex may correspond to retinal loci of different rod-cone proportions with different neural relationships (*v. s.*). This fact is probably the main reason for the finding that two points in the visual cortex explored simultaneously, demonstrated different spectral sensitivity patterns, even when diffuse monochromatic retinal illumination was used (p. 60—61).

SUMMARY: *The spectral absorption of the preretinal media, as well as the reflective properties of the tapetum, has been studied in the cat. Its mixed retina contains receptor mechanisms with broad band spectral sensitivity (scotopic and photopic "dominators" with maxima at about 500 and 560 mμ respectively) as well as narrow band mechanisms ("modulators"). In the cat, the modulator mechanisms often require indirect methods to be demonstrated. Certain results from investigations of the ERG and single retinal elements indicate a heterogenous nature of the scotopic mechanism. This would then be responsible for deviations of the retinal sensitivity from the rhodopsin absorption curve in the scotopic state. Intensity of illumination is reflected in absolute frequency patterns of discharge on single optic nerve fibres, and a specific frequency modulation of the central*

messages for various colours has also been demonstrated, as well as differences in conduction velocity for different colours.

Electrical stimulation of the central visual pathways has made certain conclusions possible as to the pre- and post-synaptic origin of certain components of evoked potentials in the LGN and visual cortex. These conclusions cannot at present be applied to the many different types of responses recorded in the present experiments by surface leads in these structures.

B. PREVIOUS STUDIES OF CEREBRAL RESPONSES TO COLOUR

There is a remarkable contrast between the abundant literature on colour perception and the factual knowledge of central nervous potential phenomena following retinal illumination with spectral light.

The attractive hypothesis of LE GROS CLARK (1949) which implied a laminar distribution of colour messages in the different layers of the LGN has not been supported by later anatomical work (CHOW 1955), and there is no physiological evidence to support it. Thus, COHN (1956 a and b), working with microelectrode technique in the LGN of the cat, did not find a laminar colour distribution in this structure. Neither did he find evidence of restricted spectral sensitivity. In the monkey, moreover, it has recently been demonstrated that single LGN cells may show narrow band spectral sensitivity in all principal layers (DE VALOIS, SMITH, KITAI and KAROLY 1958) and the theory of LE GROS CLARK has therefore been disproved.

Cortical responses to colour were studied by ADRIAN (1946) who made fundamental observations on rod and cone components in the ERG. In the striate area of the monkey he found responses to colour to be much more pronounced when the photopic ERG was present. In macular cortical fields the response to blue light was often absent while responses to red light were large (*v. s.*, p. 10).

In the cat, MOTOKAWA and his coworkers (1953) who had previously (1952) demonstrated colour sensitive retinal mechanisms in the same species, studied cortical responses to monochromatic flicker, *i. e.* with a method in principle similar to the one used here. They were unable to find significant

deviations from the scotopic luminosity curve in the spectral distribution of amplitude. With intense monochromatic light, however, some differences in wave forms could be recorded in responses to different colours. Although it was assumed that these differences were chiefly due to "differences in luminosity", they expressed the hope of "the appearance of some new means of analysis of visual cortical responses".

The studies of LENNOX and MADSEN (1955; MADSEN and LENNOX 1955) and of LENNOX (1956, 1958 a and b) in the cat are of immediate concern to present investigation. LENNOX and her coworker demonstrated that in the striate area responses to blue light were larger than to green light when the stimuli evoked ERG responses of equal amplitude. Certain differences in responses to colour were also noted between anterior and posterior parts of the visual cortex. When the coloured light flashes were adjusted to give equal LGN latency, the latency of cortical responses to red was shortest, longer to yellow and longest to blue and green stimuli. Recently, LENNOX (1958 a and b) studied the conduction velocity of impulses in optic tract fibers evoked by coloured light flashes of equal energy, as well as by electrical stimulation of the optic nerve. Slower fibres of the On-type responded more actively to blue than to red flashes, while faster fibres gave short latency responses to red light. The majority of Off-fibres found were not sensitive to colour, but only to brightness. In a general way these results support the hypothesis that differences in conduction velocity may also be a possible mechanism by which visual centres are informed about colour (*cf.* DONNER 1950, CHANG 1952).

Thus, the studies of LENNOX and her associate have clearly established that certain differences due to the colour of visual stimuli of equal energy may be recorded in cerebral visual centres of the cat. These results do not, however, give any information about the spectral distribution of such differences nor of the relationship between retinal and cerebral spectral sensitivity and their dependence upon the state of adaptation.

C. SCOPE OF THE PRESENT INVESTIGATION

1. The development of a technique by which spectral sensitivity could be measured in the visual centres of the brain of the cat according to principles used previously for retinal mechanisms in the same species.

2. The investigation of the projection to central visual structures of retinal receptor mechanisms with broad and narrow band spectral sensitivity including a study of the influence of dark and light adaptation upon this projection.

3. A study of the extent of such a projection to the main visual centres of the brain with special consideration of differences between the cortical ("neovisual") and brain stem ("paleovisual") afferent inflow.

4. A comparison between known retinal mechanisms for light reception in the cat and the spectral sensitivity patterns found in the visual centres.

Chapter II

Methods

CHOICE OF PREPARATION

The experiments required very stable preparations. Several anaesthetics (Nembutal, Chloralose, Dial, Urethane) were tried in preliminary experiments, but difficulties were encountered in keeping the level of anaesthesia constant. Changes in the latter resulted in the occurrence of eye movements and probably also in variations in retinal sensitivity during the longlasting experiments. Avoidance of anaesthetics has been proved to enhance the "spontaneous" activity of retinal neurons and to lower the threshold (GRANIT 1945, 1955 a, BARLOW, FITZHUGH and KUFFLER 1957). For these reasons, the investigation was carried out in non-anaesthetized preparations of the *cerveau isolé*-type (BREMER 1935). Adult cats of both sexes were used. There were 21 experiments satisfactory enough to be included in the final analysis. In the remainder (about 20), the measurements of the records showed that insufficiently stable conditions had been present.

OPERATIVE PROCEDURES

The cat was anaesthetized with ether and an intravenous cannula was inserted into the femoral vein. The operation was then carried out under a short acting barbiturate (Pentothal) which was administered in doses of 12.5 mg in 0.5 ml saline, when there were signs of awakening. The head was rigidly fixed in a special head-holder which permitted access to occipital parts of the brain. Through an occipital craniotomy on the left side a small temporo-occipital resection was carried out by suction during which care was taken not to injure the

optic radiation or the visual cortex. The cortical resection exposed the left side of the brain stem above the tentorium. A final dose of Pentothal was then given. Immediately afterwards, during the period of Pentothal apnoea, the brain stem was sectioned by means of a thin steel spatula. This was introduced through the lateral border of the left posterior colliculus. It was then carried perpendicularly down, to the left and right side to meet bony resistance around the brain stem. The plane of section was situated approximately 4 millimeters posterior to the interauricular plane (*i. e.* at stereotaxic plane minus 4). The bleeding was usually very scant and easily controlled by means of small pieces of Gelfoam. It is believed that the dose of Pentothal immediately preceding the brain stem section, by lowering the blood pressure helped to avoid bleeding. Within 10—20 minutes the preparations usually recovered and demonstrated good reflexes and a slow regular breathing (*cf.* BREMER 1935). With the technique used, brain edema was infrequent. During the experiments the cat's temperature was held close to $+38°C$ by heating devices.

The right visual cortex was then exposed and the dura opened in a pool of heated mineral oil which was kept at body temperature throughout the experiment. When recording from the LGN or the superior colliculus these structures were exposed by acute occipital cortical removals bilaterally and after haemostasis covered with mineral oil. In three experiments the entire visual cortex (including the suprasylvian gyrus) had been ablated about one year prior to the final experiment in which collicular and LGN responses were investigated.

The left eye was rigidly fixed by conjunctival sutures to a metal ring which by its shape was held in place by the eyelids, and by some sutures to the head holder. In most experiments, the nictitating membrane was left intact and prevented from protruding by the metal ring. The pupil of the left eye was maximally dilated at the start of the operation by a few drops of 1 % atropine solution, or by placing a small pellet of atropine sulphate in the conjunctival sac. Occasionally it was necessary to inject about 0.25 ml of a 1 % atropine sulphate solution into the conjunctiva. During the experiment the cornea was pro-

tected from drying with Ringer's solution. The right eye was closed by sutures between the eyelids. The head was arranged so that no light fell on the right side of it.

Light stimulation was carried out with the cat covered with black cloth placed in a box painted black into which the narrow light beam from the monochromator was directed from above by means of two prisms. Light reflection was diminished in the laboratory by means of black curtains and wall paint and by the use of a black coat by the experimenter.

Once the cortical and eye electrodes had been tested, the cat was left in the black box to recover fully from the anaesthesia and to darkadapt for a period of at least one hour (as a rule 1.5 hours) before any light stimulation was carried out.

OPTICAL SYSTEM

Tungsten strip filament lamps were used as light source. The filament had a width of 2 millimetres and a length of 10 millimetres. The lamps had been calibrated by the manufacturer (*Philips*) at a mean colour temperature of 2800°K. Current was supplied by a large storage battery and its constancy was carefully checked during the experiments by an ammeter.

Through a condensor system of apochromatic lenses the light was focused upon the collimator slit of a *Hilger-Tutton* monochromator (GRANIT and MUNSTERHJELM 1937). At a short distance in front of the monochromator a rotating sectored disc was placed giving intermittent interruptions of the light beam with a light to darkness ratio of 1:1. The disc was driven by a Velodyne motor with electronic negative feed back through which the set rate of revolutions was held extremely constant (ENROTH 1952).

In the narrow light beam in front of the monochromator, a photocell was placed which triggered the oscilloscope beam. Signals from the photocell indicating On and Off could also be recorded on a separate beam. Neutral density filters (*Ilford*) for stepwise intensity variations, a neutral wedge (for continuous variations over about 1 log unit density), and a filter for long wave-lengths (*Calflex Wärmefilter* with very steep absorp-

tion gradient above 6700 Å) were also used. The spectral absorption was determined for all filters and wedges used by means of a *Unicam* spectrophotometer.[1]

The position of the monochromator prism was calibrated several times during the investigation by means of a Mercury arc lamp focused upon the collimator slit (opening 0.1 mm). The exit slit was adjusted to let through 20 mμ at wave-length 435.8 mμ and 30 mμ at 623.4 mμ whilst observing it with an eyepiece. From the exit slit of the monochromator the light beam was conducted through a condensing lens and two prisms to fill the whole dilated pupil of the cat's eye. The distance from the lens to the cat's cornea was about 25—30 cm. Since the cat's eye is hypermetropic a very large area of the retina was diffusely illuminated in this fashion.

Careful measurements of the relative spectral energies were carried out for each lamp used by means of a *Hilger-Schwarz* vacuum thermopile model FT 12 in conjunction with a *Siemens* Super Galvanometer. The deflection of the galvanometer was optically amplified 15 times. From the values obtained for the wedges and filters, curves were plotted for gradient density in each wave-length used for the final calculations which were all made in terms of relative quantum intensity (DARTNALL 1953, 1957, GRANIT 1955 a). No correction was made for preretinal absorption and tapetal reflection (*v. s.* and p. 80).

It is essential to point out that the energy available from the optic system was comparatively limited and that light stimulation was carried out with small intensities in general. At the lowest intensities at which cerebral responses were obtained in the dark adapted preparations, *i. e.* at "cortical threshold", a fully dark adapted human observer could not clearly distinguish colours when looking directly into the monochromatic light beam. Nevertheless, it is possible by the *Hilger-Tutton* monochromator (used also by GRANIT in his work) to record photopic dominators in terms of single-fibre responses from light

[1] The author is indebted to Mrs. E. Dodt for carrying out these measurements.

adapted eyes. The intensity available therefore reached well into the cone range of thresholds.

RECORDING TECHNIQUE

Responses from the cortex, from the exposed lateral geniculate body, or from the superior colliculus were picked up by small platinum ball electrodes (diameter 1.5 millimetres) suspended on a thin insulated spiral wire. Usually only the On component, but sometimes also the Off, was recorded. An indifferent electrode was usually placed on the neck muscles. Control studies were made with "bipolar" cortical leads, 5—8 millimetres apart. The signals were fed to condensor coupled amplifiers (Tektronix, type 122 preamplifier, in combination with a four stage DC-amplifier) and a two tube, four beam cathode ray oscilloscope. The preamplifiers were usually used with a high frequency cut off filter at 10 Kc. By different input capacitances the time constant could be varied between 0.5 and 0.02 seconds. Usually 0.2 or 0.02 was used. This limitation of the frequency response of the recording system was found necessary in order to eliminate some of the high voltage slow waves of the background EEG activity characteristic of the *cerveau isolé* preparation.

In spite of the restricted frequency response of the amplifiers the signal-to-noise ratio was unfavourable, especially for small responses in central visual structures. A photographic superimposition technique (DAWSON 1954) was therefore used throughout the investigation for recording the responses from the oscilloscope (Fig. 5 and 6). Thus, at least 30, generally 40, responses were superimposed on each frame. Recording was usually made with only one channel, but in some experiments two responses were studied simultaneously in two identical channels. Care was taken to develop the films (film width 6 cm) from the same experiment equally in order to obtain equal contrasts in all frames.

EXPERIMENTAL PROCEDURES

Intermittent monochromatic light stimulation was carried out with eleven or twelve wave-lengths, usually at every twentieth mμ between 430 and 630 or 650 mμ. In some experiments additional wave-lengths were used. The order of wave-lengths was chosen at random. Green light of 510 mμ was arbitrarily chosen as a reference wave-length. It is realized that this reference point has given some of the records an unconventional form.

Initially, in each experiment (after dark adaptation for not less than one hour) the retinal spectral sensitivity was determined according to the 'resonance' method of GRANIT and WIRTH (1953). This method includes the calculation of a sensitivity curve from relative quantum intensity reciprocals to monochromatic light stimuli giving equal amplitudes of ERG responses to flickering light. The retinal spectral sensitivity was usually determined at a flicker frequency of 5.2 per second. At this rate, the b-wave of the ERG, being the chief constituent of the response, was of sufficient amplitude to be measured with the resonance meter. (For most of the cerebral visual responses a slightly lower frequency, 3.1 per second, was used in order to obtain larger responses; *v. i.*, p. 38). With the unanaesthetized preparation used, the ERG-responses were found to be extremely stable, (variations were less than ±5 %) and the determination of retinal spectral sensitivity did not usually last more than 30 minutes.

Without changing the experimental conditions (except the rate of flicker in most experiments), recording was then started from cerebral visual structures after an additional 10—15 minutes in total darkness. Preliminary experiments showed that it was not possible to use the resonance meter for measurements of central visual responses which often had a complex form and were mixed with the irregular background EEG activity. Early in the study, it was also found that cortical responses to monochromatic stimuli of an intensity adapted to the retinal spectral sensitivity curve were fairly similar but not identical in details (*v. i.*, p. 38 and Fig. 5).

In order to obtain more exact measurements of responses of

equal amplitude or latency, and of the development of variations in them, responses from cerebral visual structures were studied for each wave-length with stepwise increasing intensities. Thus, for each wave-length tested an "intensity series" was obtained consisting of 15 or 30 steps (intensity difference 0.17 and 0.34 log units respectively) covering about 4 log units. For every step a superimposition photograph representing 30 to 40 responses was obtained (Fig. 6). The exposure time was measured on a stop watch (usually 10 seconds). Every series was started at a low intensity which was usually 1.0 to 0.5 log units below "threshold", *i. e.* the lowest intensity at which the cortical responses could be traced in the records. The smaller responses were never discernible in single sweeps of the oscilloscope. The threshold was as a rule found to be 1 to 2 log units below the intensity level at which the retinal determinations were made. Between the steps, when the intensity was increased on the wedge, the light stimulus was left on. The order of the intensity series was randomly varied through the spectrum and between the experiments. For the reference wave-length 510 mμ, three or more intensity series were usually obtained, *i. e.* every third or fourth intensity series consisted of a control (Fig. 7 and 8). Between each series a period of total darkness of about 2 minutes or more was allowed to pass. Since every intensity series took 10—15 minutes, between 3 and 4 hours were required to obtain a complete set of intensity series covering the spectrum at every twentieth mμ from 430 to 630 mμ. During this time the constancy of pupillary diameter, lamp current, position of the recording electrodes, and of other conditions was checked at regular intervals.

The experiments ended by a control determination of the retinal spectral sensitivity. In some experiments retinal sensitivity values as well as cerebral visual intensity series were subsequently also obtained under conditions of moderate light adaptation (diffuse illumination with white light of 20 Lux at the cornea). An ordinary 40 watt light bulb was then lit at the opening of the black box, some 50 cm. from the cats eye. In one of these experiments a final, third set of intensity series was recorded under dark adaptation.

MEASUREMENTS OF RECORDS, CALCULATIONS AND CONTROLS

The photographic records of the superimposed cerebral responses (Fig. 5 and 6) did not permit evaluation of finer details. Therefore, only the following two parameters were measured (with sliding callipers calibrated in millimetres and equipped with a vernier): 1.) *Latency:* the distance from the onset of the sweep (coinciding with onset of the light stimulus) to the peak of the first constant (usually positive) large deflection. 2.) *Amplitude:* The largest over all peak to peak amplitude of the first constant response complex following the On or Off signal. All responses were measured three times and an average was used for the further procedures. The amplitude measurements could not be made as exact as those of latency due to the photographic technique. However, by training, a surprisingly high degree of constancy was obtained. Variation in the latency measurements did not exceed ± 3 % and in the amplitude measurements ± 10 %. Several series of records were discarded which showed larger variation than these due to unstable experimental conditions or unsatisfactory photographic technique.

Plots were then made for each wave-length of both the latency and the amplitude as a function of log relative intensity. Examples of such plots are given in Fig. 9 and 10. Due to the photographic technique every curve for each wave-length is based upon at least 500 individual responses. Intensity reciprocals to equal amplitudes, or latencies, in different wavelengths taken from these plots then formed the basis for calculation of cerebral spectral sensitivity curves. In each fully analyzed experiment several sensitivity curves were calculated at many ordinate levels for increasing amplitude or decreasing latency, *i. e.* variations in the spectral sensitivity were studied as a function of flicker intensity. As will be shown, this implies a study of changes in spectral sensitivity during advancing light adaptation in flicker.

As mentioned, values obtained for 510 mμ in repeated series were used as references and also as controls to judge the "stability" of the preparations (Fig. 7 and 8). Two simplified gra-

phical methods of illustrating "stability" have been used in the diagrams. In the *logarithmic diagrams* the average dispersion of the reference values at 510 mμ has been indicated by a perpendicular bar. The over all average dispersion was ±0.19 log units (0.17—0.62) for the equal latency diagrams and ±0.21 log units (0.19—0.58) for the amplitude diagrams.

In the *percentual diagrams* two sensitivity values for each wave-length were calculated, one in relation to the *average control* value from all the intensity series of the experiment obtained at 510 mμ (dots joined by dashed lines), and another in relation to a *"running control"* value at 510 mμ (circles), *i. e.* to the 510 mμ value closest in time during the recording period. The distance between the dots and the circles was thus large when conditions varied and small in stable preparations. It is readily seen from, *e. g.*, Fig. 28 that curves based upon one index (On amplitude) signalled great stability, while curves from another index of the same response (Off amplitude) indicated instability. This phenomenon has been interpreted to show a inherent instability of the mechanisms represented in the Off response (*v. i.*, p. 81).

On the whole, the variations due to methodological factors as well as to inherent instability of the mechanisms under study were very large. In the individual sensitivity records only deviations well outside the dispersion of the control values are significant. However, the series of sensitivity curves for a great range of increasing intensities in many experiments disclosed that even smaller humps behaved in a consistent manner. Furthermore, certain systematic changes in spectral distribution from one experiment to another could be recognized in the curves. For reasons to be discussed below (p. 82), it seems very unlikely that such changes were due to random variability. Instead, the conclusion is reached that they were caused by characteristics of the receptor-conductor system studied.

In all sensitivity curves the absorption curve of pigment 497 of DARTNALL (1953) (in the following called the "rhodopsin curve") has been indicated. It is believed that this pigment corresponds to the visual purple of the cat (GRANIT 1955 a).

SUMMARY: *In unanaesthetized cats (cerveau isolé preparations) the left eye was diffusely illuminated with monochromatic flicker of constant rate and of known energy. A tungsten filament lamp was used as a light source in connection with a monochromator. The intensity of the flicker for each wavelength tested was increased in small steps and superimposed records of 30—50 On (and Off) responses in right-sided cerebral visual centres were obtained at each step. "Intensity series" were obtained for wave-lengths at every 20th mμ from 430 to 650 mμ in random succession. Several series were obtained for the control wave-length 510 mμ. From plots of latency or gross amplitude of the responses against intensity, relative quantum intensity reciprocals were obtained for equal responses in the wave-lengths tested. Calculations were then made of spectral sensitivity, defined as intensity reciprocals (1/I) to equal amplitude or latency in the different wave-lengths. Sensitivity curves (log curves or percentual curves) were calculated at several values of falling equal latency or rising equal amplitude, i. e. spectral sensitivity variations were studied as a function of intensity of flicker, that is during advancing light adaptation in flicker. The sensitivity patterns obtained in cerebral visual centres were related to the rhodopsin absorption spectrum, as well as to the retinal spectral sensitivity of the same experiment which was measured by means of the ERG-resonance method.*

Chapter III

Results

The results will be presented mainly in the form of spectral sensitivity curves. As mentioned, the 510 mμ wave-length was arbitrarily chosen as reference and all curves have been plotted in relation to the rhodopsin absorption curve (drawn in full in both logarithmic and percentual curves; *v. s.*, p. 31, and legends to Fig. 11 and 27).

A. PRIMARY EXPERIMENTAL FINDINGS

1. *Retinal spectral sensitivity*

Fig. 1 summarizes retinal spectral sensitivity distributions obtained by 24 determinations in 15 experiments under conditions of dark adaptation according to the method of GRANIT and WIRTH (1953; *v. s.*, p. 29). In 8 of the experiments two determinations were made, one before, and one after recording of the cerebral intensity series. In these cases there was a fairly close agreement between the two curves obtained, in spite of the fact that a 3—5 hour period had passed in between (Fig. 2 A and B and 3 A and C). The retinal variations recorded with the resonance method were thus much smaller than for the cerebral determinations of spectral sensitivity.

It is evident from Fig. 1 that all retinal curves demonstrated a significant deviation from the rhodopsin curve at 470 to 490 mμ. This finding confirms fully the "scotopic blue-shift" of retinal spectral sensitivity previously obtained in the cat with flicker by GRANIT and WIRTH (1953).

In several experiments another conspicuous, though smaller, deviation from the rhodopsin curve was found at 530 mμ, and, when present, this hump, like the larger blue one, also appeared

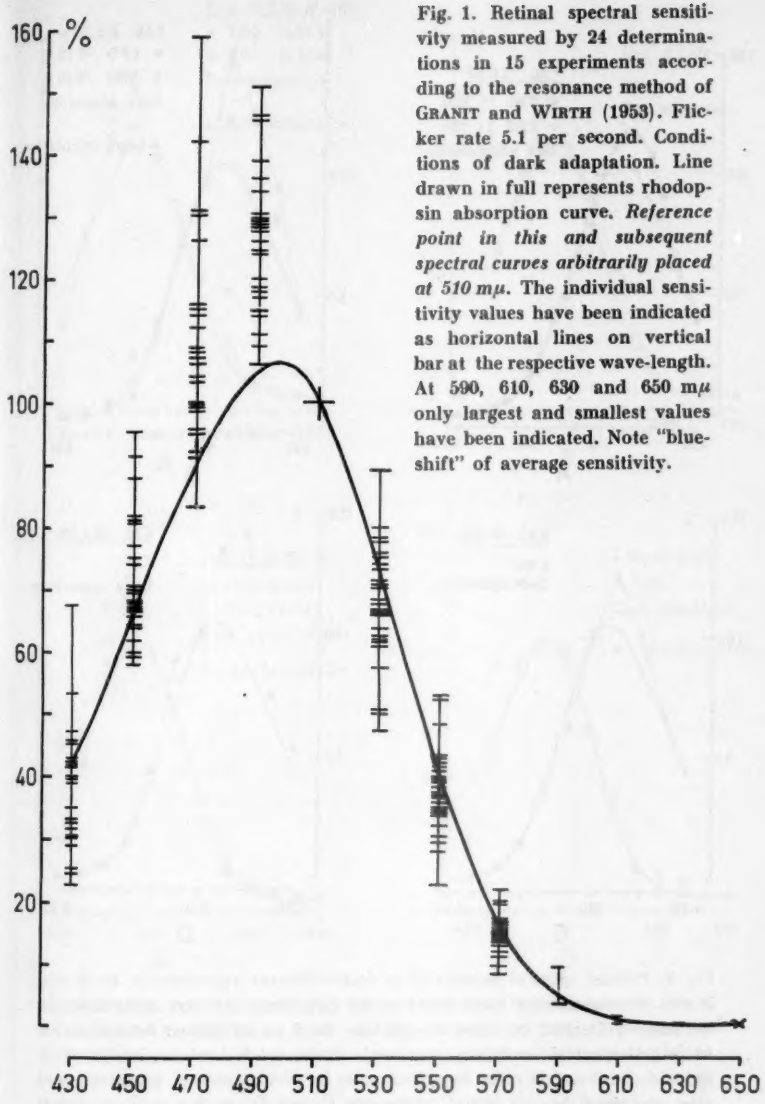

Fig. 1. Retinal spectral sensitivity measured by 24 determinations in 15 experiments according to the resonance method of GRANIT and WIRTH (1953). Flicker rate 5.1 per second. Conditions of dark adaptation. Line drawn in full represents rhodopsin absorption curve. *Reference point in this and subsequent spectral curves arbitrarily placed at 510 mμ.* The individual sensitivity values have been indicated as horizontal lines on vertical bar at the respective wave-length. At 590, 610, 630 and 650 mμ only largest and smallest values have been indicated. Note "blue-shift" of average sensitivity.

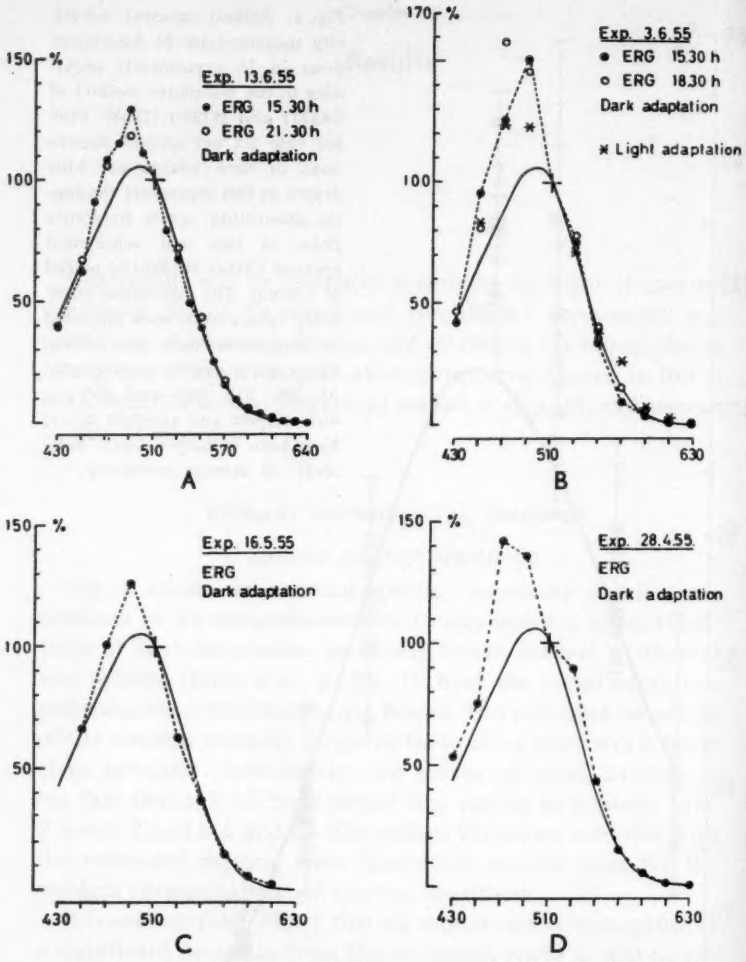

Fig. 2. Retinal spectral sensitivity in four different experiments. In A and B two determinations were made under conditions of dark adaptation at the times indicated, in C and D only one. In B an additional determination of retinal spectral sensitivity was made under modest adaptation to white light. A, B, C and D refer to experiments in which spectral sensitivity was also measured in the visual cortex, in C and D in two cortical points simultaneously. See text, p. 34 and 38. Curves drawn in full represent rhodopsin absorption.

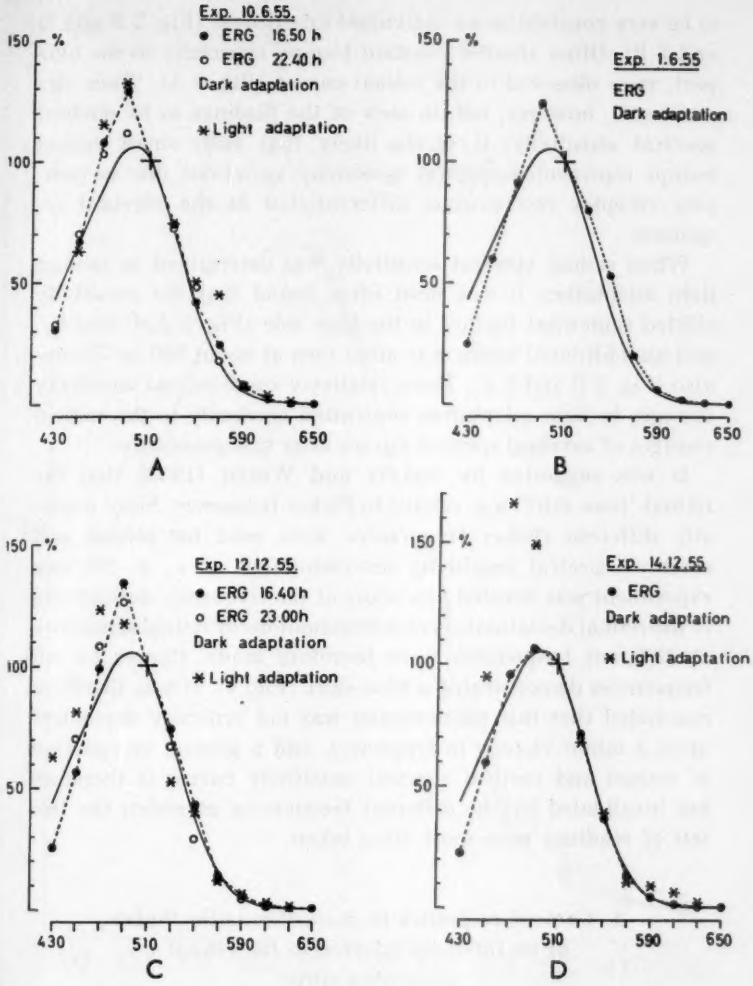

Fig. 3. Retinal spectral sensitivity in four different experiments. In A and C two determinations were made under conditions of dark adaptation. In A, C and D a second determination was made with additional adaptation to white light. A and B refer to experiments in which spectral sensitivity was also measured at the lateral geniculate, C and D to experiments with measurements at the superior colliculus. See text, p. 34 and 38. Curves drawn in full represent rhodopsin absorption.

to be very constant in an individual experiment (Fig. 2 B and D and 3 B). Other smaller constant humps, especially in the blue part, were observed in the retinal curves (Fig. 2 A). Their size was small, however, but, in view of the findings as to cerebral spectral sensitivity, it seems likely that such small retinal humps represented spectral sensitivity variations due to complex receptor mechanisms differentiated in the cerebral responses.

When retinal spectral sensitivity was determined in modest light adaptation, it was most often found that the sensitivity shifted somewhat further to the blue side (Fig. 3 A, C and D), and an additional hump was often seen at about 550 to 570 mμ also (Fig. 2 B and 3 A). These relatively small retinal sensitivity changes in light adaptation contrasted markedly to the radical changes of cerebral spectral curves after this procedure.

It was suggested by GRANIT and WIRTH (1953) that the retinal "blue-shift" was related to flicker frequency. Since normally different flicker frequencies were used for retinal and cerebral spectral sensitivity determinations (*v. s.*, p. 29) one experiment was devoted to a study of the frequency dependence of the retinal deviations. Five determinations of retinal sensitivity at different frequencies were therefore made. Curves for all frequencies demonstrated a blue-shift (Fig. 4). It was therefore concluded that this phenomenon was not critically dependent upon a minor change in frequency, and a general comparison of retinal and cortical spectral sensitivity curves is therefore not invalidated by the different frequencies at which the two sets of readings were most often taken.

2. *Cortical responses to monochromatic flicker of an intensity adapted to the retinal sensitivity curve*

In several of the initial experiments, stimulation was carried out under conditions of dark adaptation with monochromatic flicker of an intensity adapted to the retinal sensitivity curve, *i. e.* with light stimuli which evoked an ERG response (mainly b-wave) of equal amplitude in different wave-lengths. When

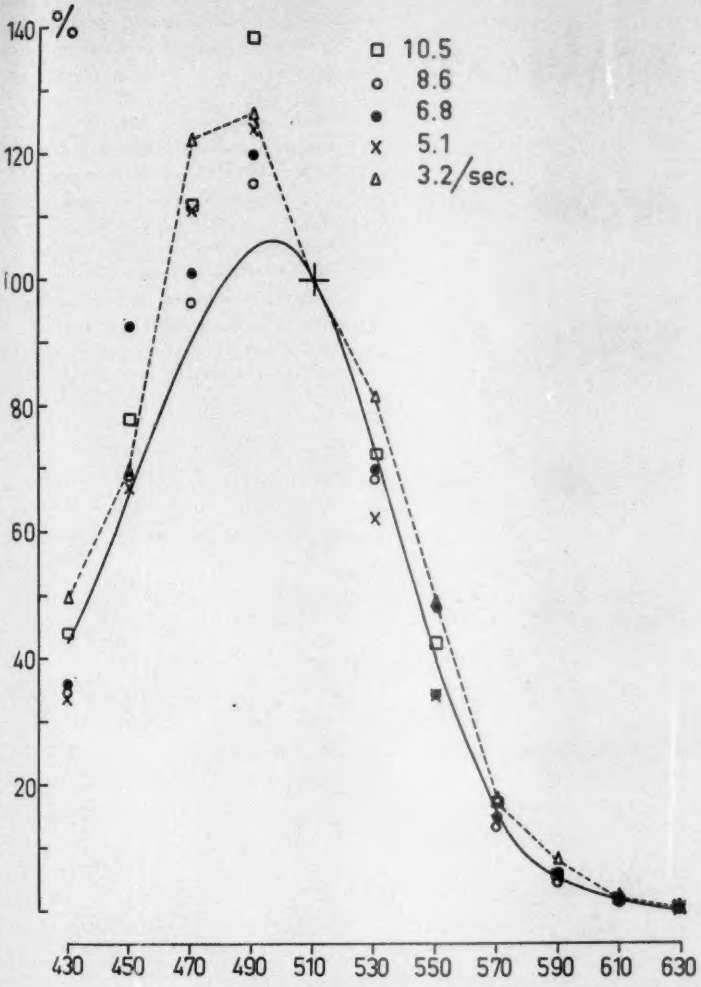

Fig. 4. Retinal spectral sensitivity measured at five different rates of flicker in the same experiment. Symbols for different flicker frequencies indicated in upper right corner. Line drawn in full represents rhodopsin absorption curve. Dotted line drawn between points refers to frequency 3.1 per second which was the one used in most determinations of cerebral spectral sensitivity. Most retinal determinations were made with a flicker rate of 5.1 per second. Note presence of 'blue-shift' of the peak at all frequencies. See text, p. 38.

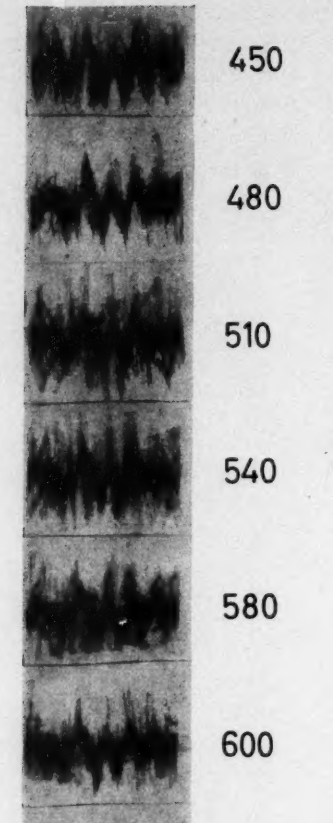

Fig. 5. Superimposed records of 30—40 On responses to monochromatic flicker at a point in the visual cortex. The intensity of the illumination with seven different wavelengths (450—620 mμ) was adapted to the spectral sensitivity curve of the stimulated eye as measured from the ERG. (See text, p. 41.) Note the gross similarity between the responses, but that smaller differences in the amplitude as well as latency are apparent on more detailed comparison. Width of original records was 60 mm.

found that they often resembled each other throughout the spectrum. However, on careful inspection of the superimposed records several smaller differences in form, amplitude and latency could be detected (Fig. 5). Many of these differences were accentuated by varying the order of wave-lengths or the time of exposure to the flicker. It proved, in fact, impossible to obtain exactly identical responses in the cortex in different wave-lengths by using intensities adapted to the curve of retinal sensitivity.

This finding demonstrated that the cortical sensitivity followed the retinal spectral distribution only grossly (*cf.* MOTOKAWA, TUKAHARA and EBE 1953), a fact confirming the many previous studies which suggested an heterogenous light reception mechanism in the scotopic state (*v. s.*, p. 16). Therefore, a procedure was worked out which allowed a more exact determination of spectral sensitivity distributions from cerebral visual responses, a procedure in which the responses to each wave-length were studied as a function of intensity of the illumination.

3. *The cortical responses*

Two typical "intensity series" of cortical responses are shown in Fig. 6. The main On and Off components are clearly seen. Repetitive cortical On responses were found in some locations at certain intensities and they were studied in special measurements.

In the great majority of the experiments, repeated recordings of control series of wave-length 510 mμ demonstrated that the technique provided a sufficient stability of the preparations to justify a comparison between different wave-lengths. This fact, of basic importance for the present investigation, is apparent in Fig. 7 and 8. These figures show four curves each obtained from different control series at 510 mμ during a period of about 3 hours. It is seen that the variations in latency are comparatively smaller than those in amplitude. This was a constant finding and it is explained by factors mentioned above (p. 31) such as greater errors of measurements and amplitude

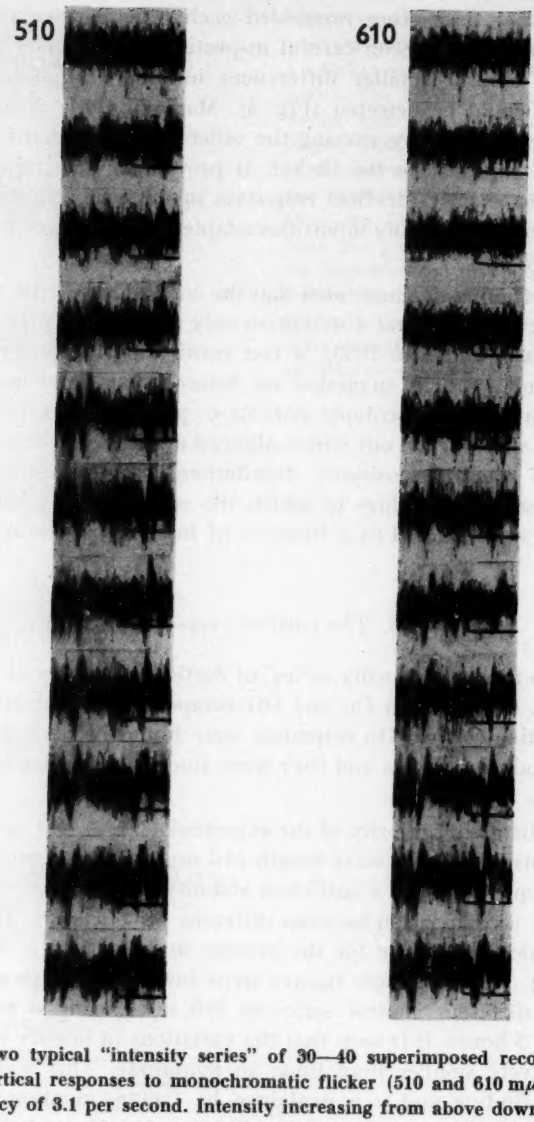

Fig. 6. Two typical "intensity series" of 30—40 superimposed records of visual cortical responses to monochromatic flicker (510 and 610 mμ) with a frequency of 3.1 per second. Intensity increasing from above downwards in eleven steps covering about 3 log units. Lower beam on each frame indi-

cortical responses to such stimulation were recorded, it was modulations by the EEG. A discussion of the "stability" of the mechanisms studied is given in Chapter IV (p. 81).

The plots of latency, or amplitude, as a function of relative intensity demonstrated smaller or greater differences in gradients between the wave-lengths tested. The latency plots, derived from more exact measurements, were very often almost linear, although smaller shifts of gradients were also seen in them. The amplitude curves, on the other hand, usually showed an initial rising phase with increasing intensity from the "threshold" (p. 27) with smaller variations in slope between the different wave-lengths. At a certain intensity there was usually a decrease of the slope of the curves for all wave-lengths. They flattened out and sometimes the slope became negative. Finally, in the highest intensities there was in most experiments a final increase of amplitude again. Examples of such latency and amplitude curves from six intensity series in one experiment (3.6.55) are given in Fig. 9 and 10. For the final calculations each individual curve was smoothed out, *i. e.* the discontinuities due to shifts of neutral filters (dotted in Fig. 7 to 10) were disregarded. It is evident that sensitivity curves can be calculated for several values of equal amplitudes in different wave-lengths on the initial rising phase, as well as on the following falling phase.

It seemed a fair assumption that the diminution of amplitude in all wave-lengths at a certain level of intensity of illumination (as well as the second phase of increase in some curves)

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cates period of illumination with beam going up when light went on. Only the main deflection of On and Off responses was measured. The *latency* of both On and Off responses were measured in msec from onset of illumination (same as onset of sweep) to the peak of the main down-going deflection, and, for the Off latency, the time of illumination was subtracted. The *amplitude* of On and Off responses were measured with sliding callipers with a vernier directly from the films (width 60 mm) from blackest part of down-going peaks to blackest part of up-going following peak. If this second peak was of a complex form, the highest component was used. The responses were recorded by small platinum ball electrodes (diameter 1.0 mm) in a point in the anterior visual area. Input capacitance of recording amplifier 0.02 seconds.

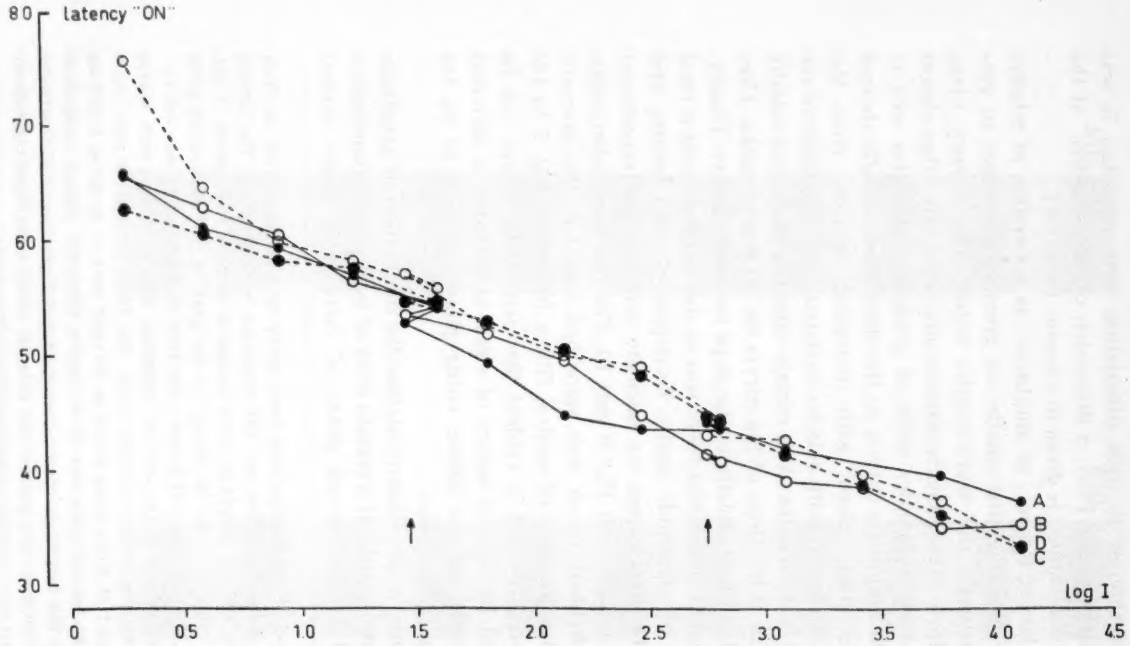
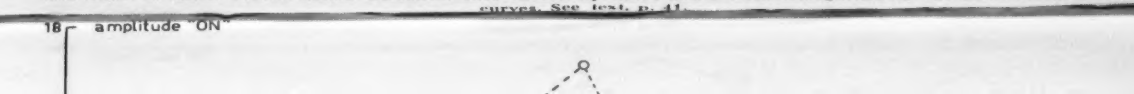


Fig. 7. Exp. 3.6.55. Conditions of dark adaptation.

Latency in msec of visual cortical On responses plotted against intensity of flicker. A, B, C and D refer to four intensity series recorded with the same wave-length (510 mμ) at about equal time intervals during 3 hours. Arrows indicate shifts of main neutral filters. Discontinuities caused by these shifts were disregarded in the final analysis of curves. **See text, p. 41.**



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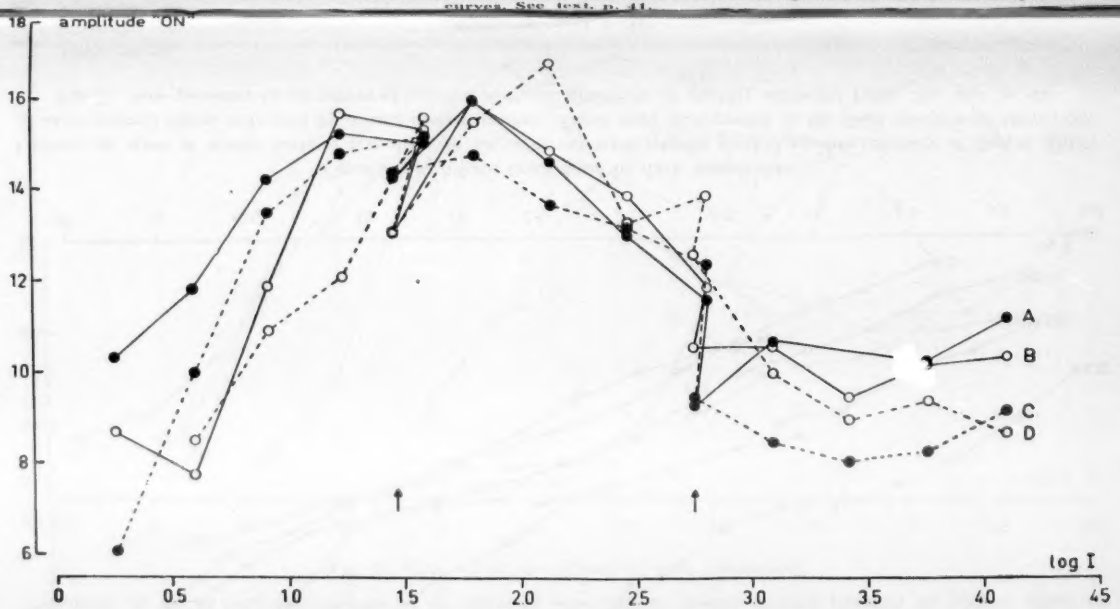

Fig. 8. Exp. 3.6.55. Conditions of dark adaptation.

Amplitude of visual cortical On responses plotted against intensity of flicker (*cf.* Fig. 7). Arrows indicate shifts of main neutral filters. Discontinuities caused by these shifts were disregarded in the final analysis of curves. Note typical initial up-going phase of amplitude when the intensity of flicker was raised. There was then a shift to a down-going phase which finally changed into a second up-going phase (best seen in curve C). Such shifts of amplitude gradient with increasing intensity were observed for other wave-lengths too (*cf.* Fig. 10). See text, p. 41.

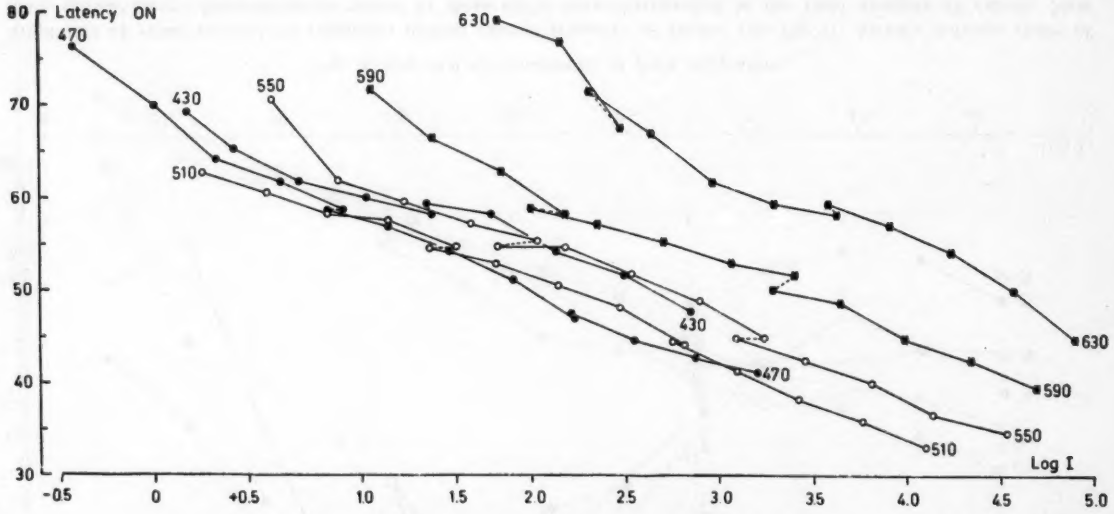

Fig. 9. Exp. 3.6.55. Conditions of dark adaptation.
Latency in msec of visual cortical responses in six different wave-lengths plotted against intensity of flicker. Shifts of main neutral filters indicated by dotted discontinuities. These were disregarded in the final analysis of curves (*cf.* Fig. 7). Note presence in all curves of smaller shifts of gradients of slightly different form. See text, p. 43.

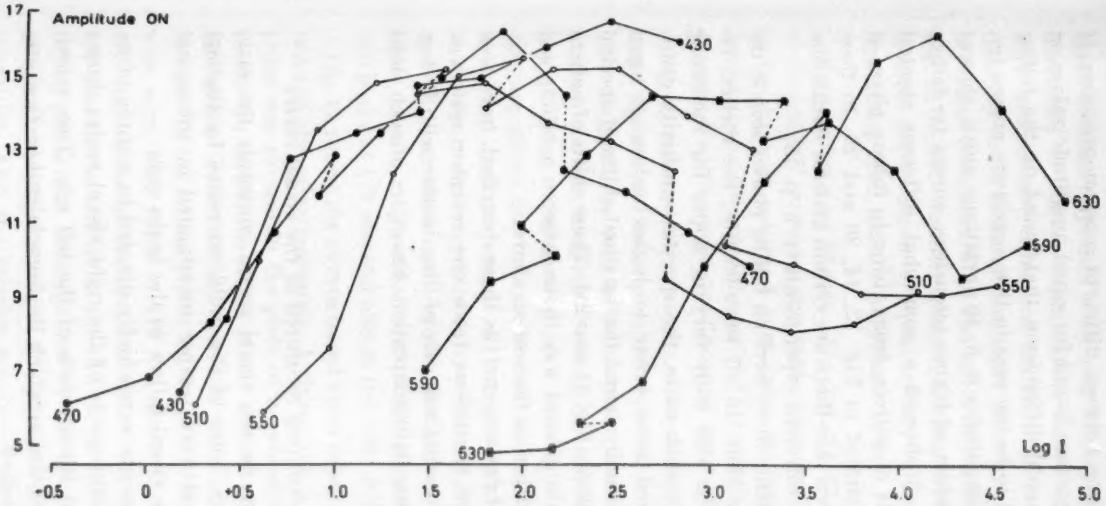

Fig. 10. Exp. 3.6.55. Conditions of dark adaptation.

Amplitude of visual cortical responses in six different wave-lengths plotted against intensity of flicker. Shifts of main neutral filters indicated by dotted discontinuities. These were disregarded in the final analysis of curves (*cf.* Fig. 7). Note presence of up-going phase of amplitude with increasing intensity of flicker in all wave-lengths. There is then (except for 430 mμ) a down-going phase, and, for 510, 550 and 590 mμ only, an indication of a second up-going phase. When calculations of sensitivity were made, an ordinate level was chosen (*i. e.*, equal amplitude), and the corresponding intensity values for the different wave-lengths were read off. Sensitivity curves were calculated separately from both the up-going and the down-going phase. In similar curves the spectral sensitivity distribution was found to change pattern from the rising to the falling phase. **See text, p. 43.**

was due to interaction between different receptor processes. If so, the sensitivity pattern found for equal amplitude values on the rising phase would differ from that found on the falling phase. This proved to be the case in that sensitivity curves for, *e. g.,* equal rising amplitudes 6, 8, 10 (arbitrary units) showed great similarities, while, at higher intensities, curves for falling amplitudes 10, 8, 6 showed a somewhat different spectral pattern. Such shifts of pattern from rising to falling phase of amplitude are illustrated in Fig. 12, 14, 26 and 28. In these figures it is also seen that there are certain general similarities in the shifts in two different experiments (*v. i.,* p. 75).

In other experiments there were no falling phases seen in the amplitude-intensity plots. In all wave-lengths, the cortical responses increased in size with varying slopes for increasing intensity. When, in such cases, the spectral sensitivity distribution was followed from lower to higher values of equal amplitude, it was usually found that gradual shifts of spectral pattern also took place (Fig. 11 and 25). These shifts of pattern often resembled in a general way those between a falling and a rising phase described in the section above.

Shifts of spectral pattern, such as those described, have been interpreted to show transitions from one receptor system to another during increasing intensity of illumination with flicker, *i. e.* during advancing light adaptation. Examples of such shifts will now be given.

4. *Spectral sensitivity measured at the visual cortex*

Results pertaining to the visual cortex constitute the main part of the findings. Some of the problems raised by findings at the cortical level were further investigated in subsequent experiments on the visual inflow to the brain stem.

Fourteen experiments were chiefly devoted to a study of responses in the anterior parts of the right visual cortex during diffuse intermittent illumination of the left eye. Two experiments were unsuccessful in which the homolateral cortical projection was also studied.

In general, the spectral sensitivity curves calculated from

small cortical responses with long latency (*i. e.* evoked by low intensities of flicker illumination under conditions of initial dark adaptation) coincided better with the rhodopsin curve than did those from larger, short latency, responses. This fact is evident from several of the series of logarithmic curves (*e. g.*, Fig. 11 to 14). Another general finding was that the series of spectral curves based on equal latency values showed a greater uniformity than did the amplitude series.

Experiment 13.6.55. (Fig. 2 A, 11, 12 and 26). Recording of On and Off responses was made from a point 15 mm posterior to the coronal suture, 4 mm from the midline on the right lateral gyrus. 16 intensity series (4 controls) were obtained in 13 wave-lengths under conditions of dark adaptation.

Fig. 11 demonstrates two sets of logarithmic sensitivity curves based upon equal *latency* values, reading from below upwards. This diagram, as well as subsequent ones of the same type, pictures changes in spectral sensitivity during increasing intensity of flicker illumination of a constant frequency. The left diagram shows the On curves, and the right the Off curves. It is evident that important differences exist between the two series. Both series grossly follow the rhodopsin curve (drawn in full), but there are regular deviations which, in the On series, show a single maximum at 490 mμ and in the Off series two peaks at 470 mμ and also in the 530—570 mμ region.

Fig. 12 shows the curves based upon equal On and Off *amplitudes* of the same responses. A rising and a subsequent falling phase was present in the plots of amplitudes against intensity. In the On series, the rising phase showed a broad peak in the blue-green (470—490 mμ) as well as a marked deviation at 530 mμ. In the falling phase, on the other hand, several changes were seen. The value at 470 mμ diminished and the peak at 490 mμ increased further and there was also an increase at 550 mμ. In the Off series the shift from the rising phase to the falling phase was accompanied by the development of a small trough at 490 mμ instead of the previous small peak at lower intensities. Also the relative sensitivity at 570 mμ decreased markedly at the highest intensities.

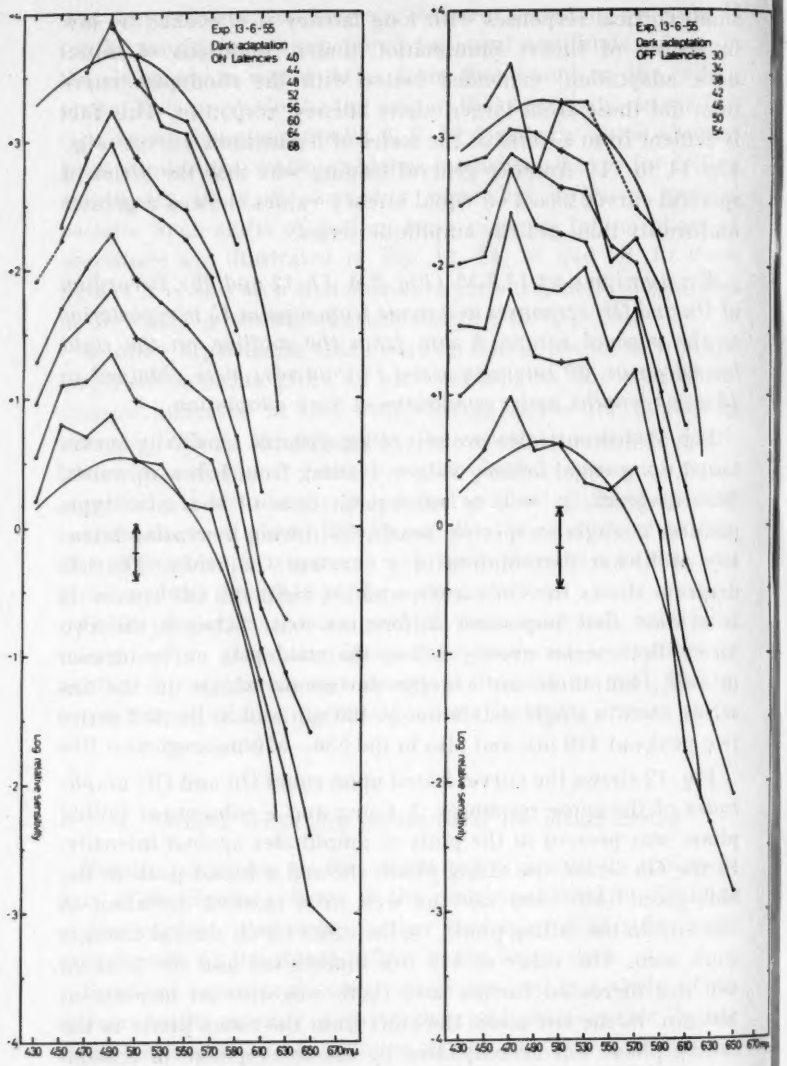

Fig. 11. Logarithmic spectral sensitivity curves calculated for values of *equal latency* of On and Off components of responses in one point at the visual cortex. Equal latency values decrease from bottom to top (see upper

The retinal sensitivity curve of this experiment is seen in Fig. 2 A.

Experiment 3.6.55. (Fig. 2 B and 13 to 17). Simultaneous recording of On and Off responses was made from a point 15 mm posterior to the coronal suture, 4 mm from the midline on the right lateral gyrus. 13 intensity series (4 controls) were obtained in 10 wave-lengths after initial dark adaptation. Adaptation to weak white light was then carried out for 1 hour, and 14 intensity series (3 controls) were obtained with the adapting light on. Finally, 10 series (2 controls) were recorded after renewed dark adaptation for one hour. This experiment illustrates reversibility of changes in cortical spectral sensitivity in dark and light adaptation.

Fig. 13 shows two series of curves based upon equal *latency* for On and Off components of the cortical responses under conditions of dark adaptation. The On series (left diagram) in this case demonstrated a much greater stability than did the Off series (*v. i.*, p. 81). In both series there was a gradual but modest increase of sensitivity in the blue part of the spectrum. Thus with increasing intensity, a peak developed at 470 mμ in the On series, while the whole blue half of the spectrum showed increasing sensitivity in the Off series (right diagram). The curves in Fig. 13 constitute some of the best examples of reproduction of the rhodopsin distribution in the visual cortex except for the modest deviation in the blue region.

Fig. 14 shows the equal *amplitude* series of the same responses. In the rising phase, the On series (left diagram) again

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right corner of each diagram), *i. e.* the intensity of illumination, or degree of adaptation, is increasing from bottom to top. Double arrow below curves in this and following diagrams indicates average dispersion of control values in several intensity series obtained at 510 mμ. Note larger variations in controls for Off series. Lines drawn through lowermost and uppermost curves indicate the rhodopsin absorption curve.

There is a general similarity between all curves calculated and the rhodopsin curve. Some deviations are, however, present which differ consistently in the On and Off series. Thus, the maximum of all the On curves lies at 490 mμ, while, for the Off series, it is to be found at 470 mμ. There is also a hump in the green (530—570 mμ) indicated in many of the Off curves. See text, p. 49.

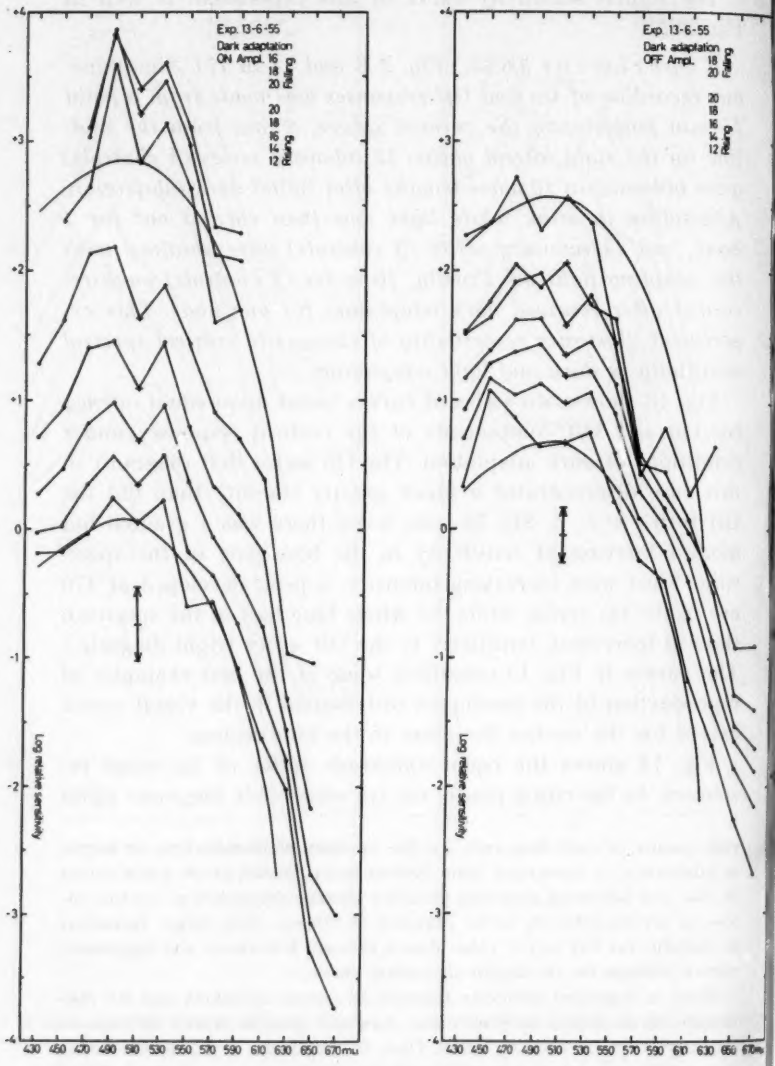


Fig. 12. See text, p. 49, and legend to Fig. 11.

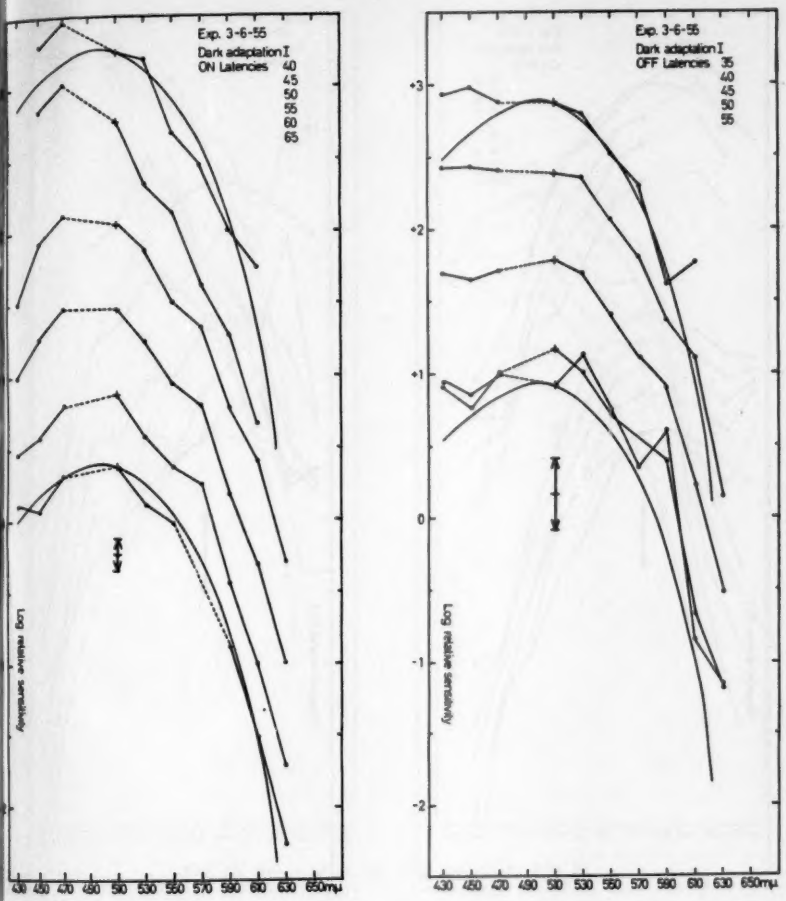


Fig. 13. See text, p. 51, and legend to Fig. 11.

showed a fairly good correspondence with the rhodopsin curve (note, however, that the 490 mμ values are lacking). In the falling phase, at higher intensities, a regular, but small peak developed at 470 mμ. In the Off amplitude series (right diagram), there was also a general increase of sensitivity in the

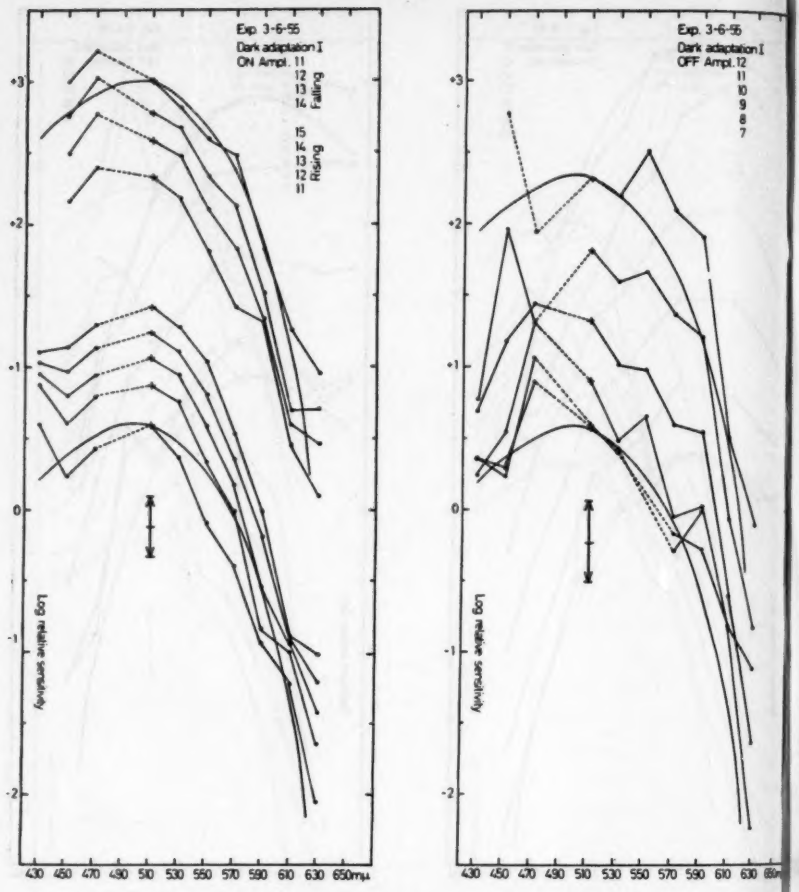


Fig. 14. See text, p. 51, and legend to Fig. 11.

530—570 mμ region with the highest intensities. Note especially the peak at 550 mμ in the uppermost Off amplitude curves.

Fig. 15 and 16 demonstrate the changes in sensitivity which occurred with weak additional *adaptation with white light*. The *latency* series (Fig. 15) for On and Off responses both showed slightly increased sensitivity in the blue (470—490 mμ) as well

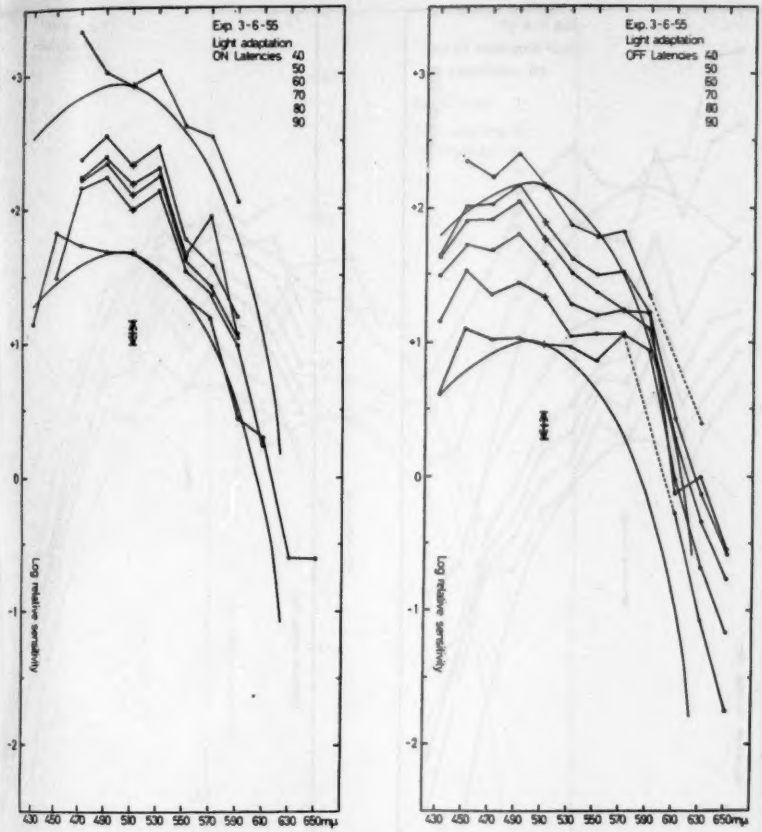


Fig. 15. See text, p. 54, and legend to Fig. 11.

as in the green region (530—570 mμ). The *amplitude* series (Fig. 16) in light adaptation showed dramatic changes in spectral sensitivity as compared to the previous measurements in dark adaptation (Fig. 14). This is most clearly seen in the On amplitude series (Fig. 16, left diagram) where all curves expressed a sensitivity distribution which, especially in the blue region, differed a great deal from the rhodopsin curve. Note the regular peak at 490 mμ, which disappeared with increasing intensity.

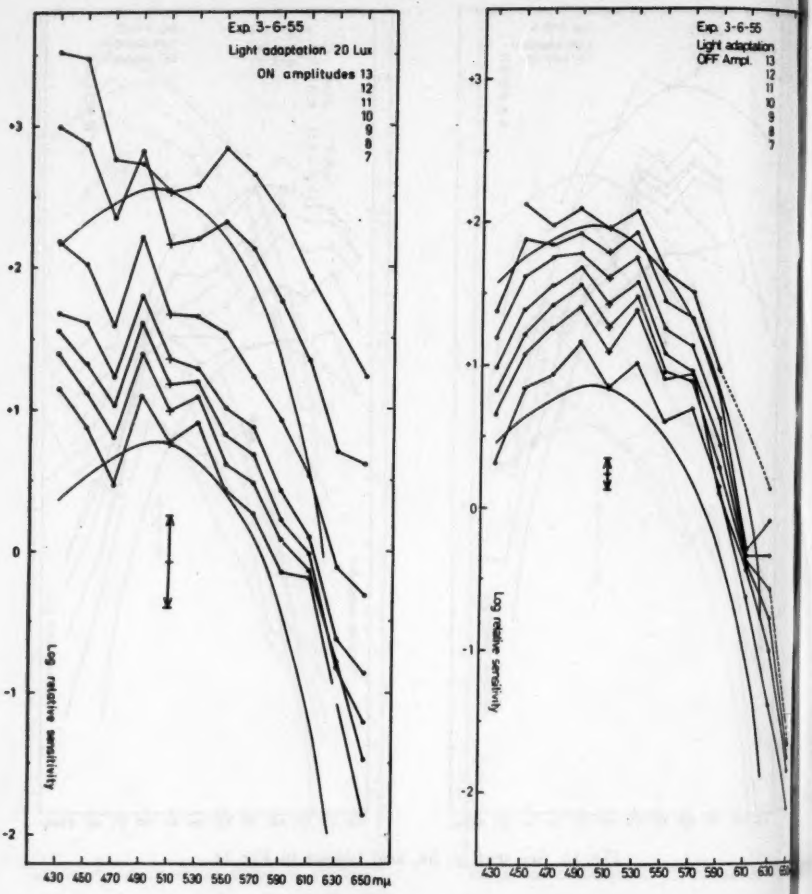


Fig. 16. See text, p. 54, and legend to Fig. 11.

Instead a large peak developed at 550 mμ together with marked increase of sensitivity in the whole red part of the spectrum. All through the series there was a remarkably high sensitivity at 430—450 mμ. The Off amplitude series (Fig. 16, right diagram) showed less striking changes but nevertheless of a kind similar to the On series.

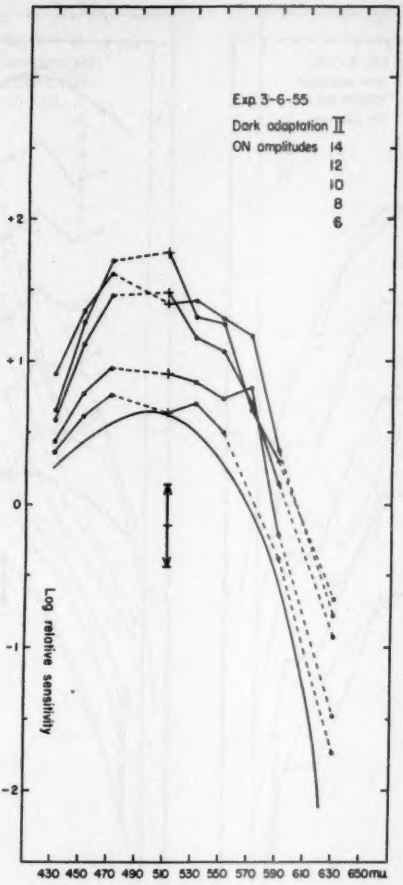


Fig. 17. See text below, and legend to Fig. 11.

In Fig. 17 one control series of equal On amplitude curves is shown which pertains to the measurements from the same cortical point after renewed dark adaptation. It is seen that the curves now agree better with the rhodopsin distribution of sensitivity and that the high peaks in the blue and at 550 mμ have disappeared (*cf.* Fig. 14 and 16 left halves).

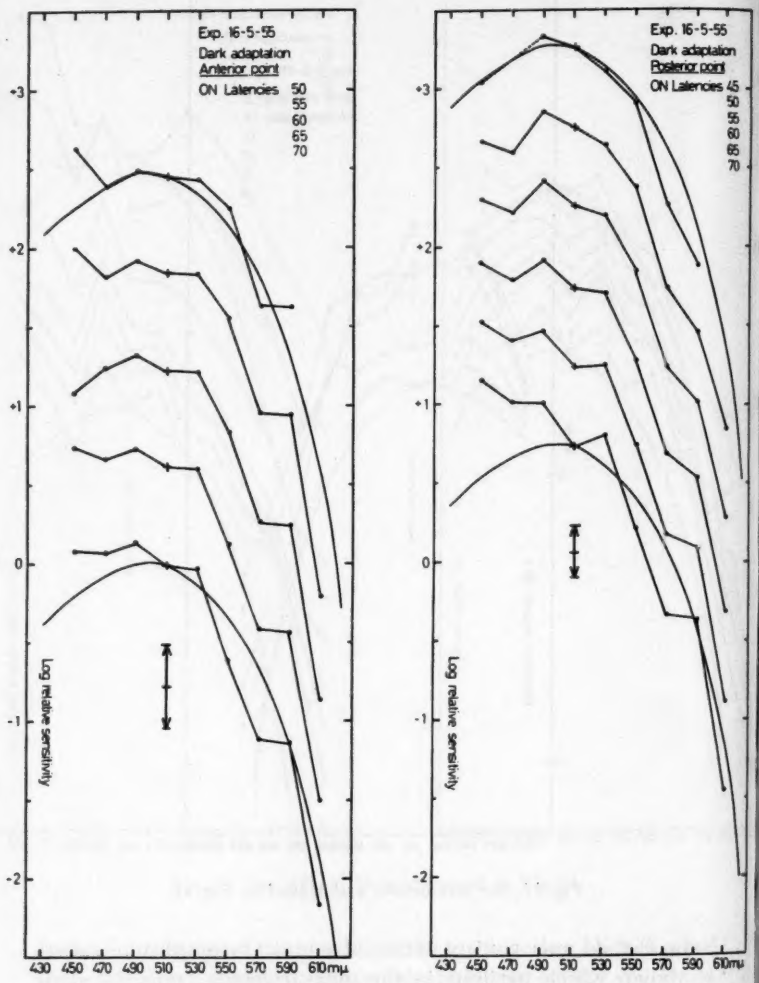


Fig. 18. See text, p. 60, and legend to Fig. 11.

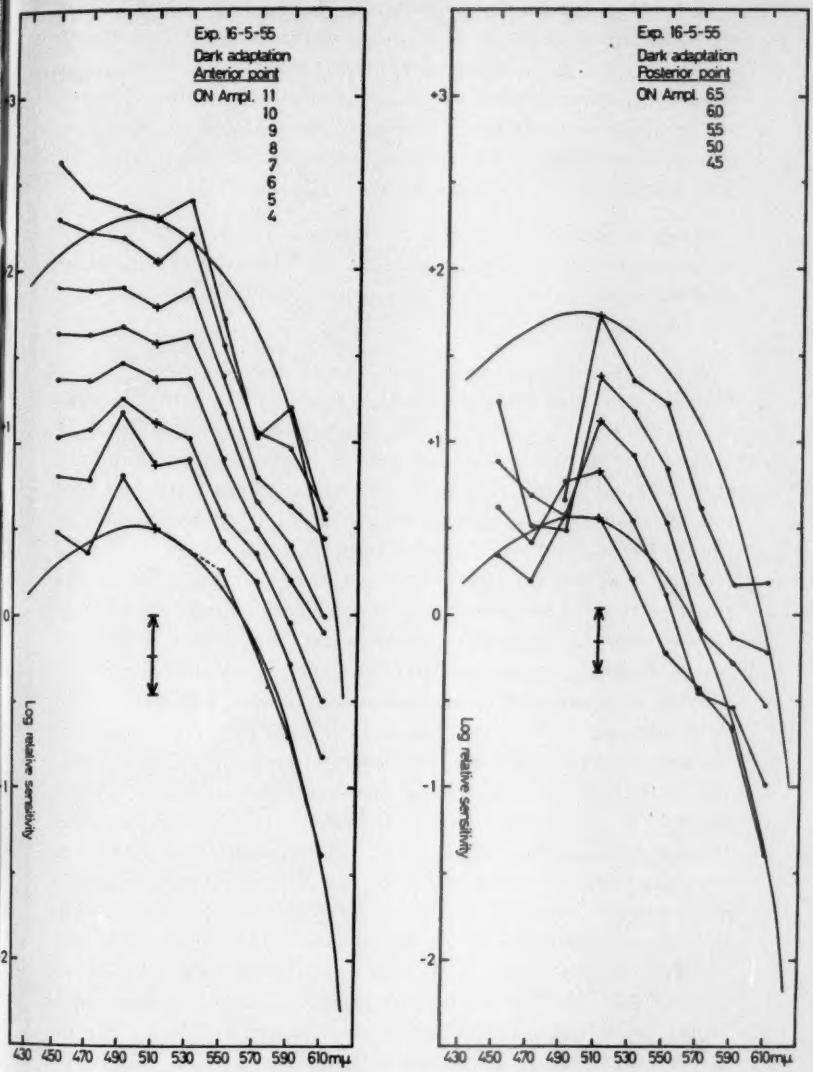


Fig. 19. See text, p. 60, and legend to Fig. 11.

In 6 other experiments, similar to the two described above, recording from single cortical points were carried out. In these, several of the above features appeared such as the systematic shift of spectral peaks associated with shifts from rising to falling phase of amplitude, and also development of increased blue and green sensitivity at high intensities of illumination or with additional light adaptation.

Simultaneous recording from two cortical points. In two experiments simultaneous recording was carried out with identical channels from two cortical points about 10 mm apart on the lateral gyrus.

E x p e r i m e n t 16.5.55. (Fig. 2 C, 18 and 19). Recording of On responses was made simultaneously from two cortical points 3 mm from the midline on the right lateral gyrus, 12 and 21 mm. posterior to the coronal suture respectively. Stimulation with 12 wave-lengths (3 controls) was carried out. The threshold for both points was found to be about equal. The responses at the posterior point were smaller than at the anterior.

Fig. 18 shows the equal *latency* curves for the anterior and the posterior On responses. The two series resemble one another closely even in showing a deviation at 450—470 mμ. On the whole, other variations are small and the curves at longer wave-lengths reproduce rhodopsin sensitivity reasonably well.

In contrast to this, the two equal *amplitude* series (Fig. 19) showed major differences between the two points. The anterior point (left diagram) showed a sensitivity which markedly resembled the one expressed by the latency of the same responses though with slightly increased sensitivity in the blue. Note also the initial peak at 490 mμ at lower intensities which was gradually replaced by small humps at 450—470 and at 530 mμ. The posterior point (right diagram), on the other hand, showed a marked trough in the 470—490 mμ region. This greatly exceeded any possible error and occurred in all curves which could be calculated. The curves calculated for the posterior point also differed substantially from the average sensitivity distribution measured in the retina in the same experiment (Fig. 2 C).

Experiment 28.4.55. (Fig. 2 B, 20 and 21). Recordings of On responses were made from two cortical points simultaneously, 4 mm from the midline on the right lateral gyrus, 10 and 20 mm from the coronal suture respectively. The threshold was lower at the anterior point than at the posterior. Fourteen somewhat unstable intensity series including 2 controls were obtained from 12 wave-lengths.

The retinal sensitivity distribution in this experiment (Fig. 2 D) showed an exceptionally high and stable value at 530 mμ. It is of interest that both sets of equal *latency* curves for the anterior and posterior points, apart from a blue peak, showed a green hump and a trough at the reference point (Fig. 20).

The equal *amplitude* curves (Fig. 21) showed a marked difference between the anterior and the posterior points in that the posterior developed low sensitivity in the green region (530—570 mμ) with increasing intensity of illumination.

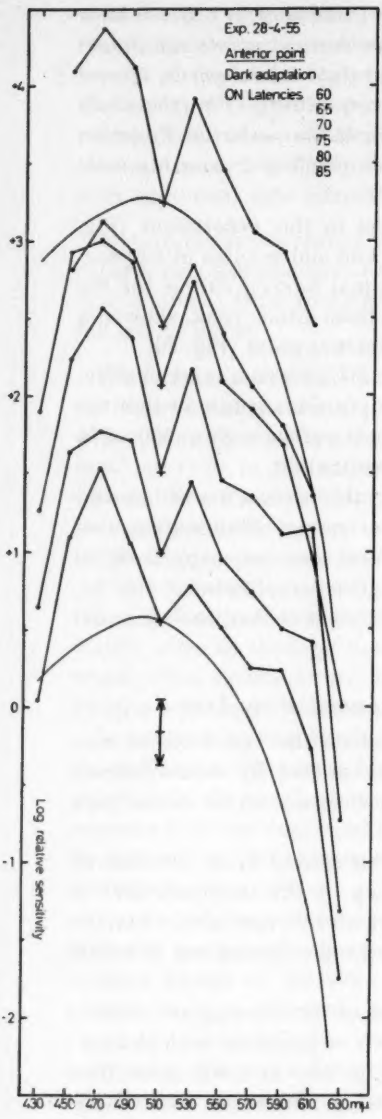
The two experiments described in this section are of interest, since they show that with diffuse retinal illumination two points in the visual cortical projection area may express different spectral distributions when gross amplitude of the responses is taken as index. The implication of this finding is discussed on p. 82.

5. *Spectral sensitivity measured at the LGN*

In three acute preparations in which the visual cortex was ablated the spectral sensitivity was studied by measurements of responses picked up by a surface electrode on the dorsal part of the LGN.

Experiment 23.5.55. (Not illustrated.) Acute ablation of occipital poles bilaterally. Recording of On responses with a electrode placed on the LGN surface on the right side. 14 intensity series (5 controls) were obtained under conditions of initial dark adaptation.

Both the plots of *amplitude* and of *latency* against relative intensity demonstrated marked shifts of gradients with increasing intensity of illumination (*v. s.*, p. 43). At lower intensities there were steep gradients for rising amplitude and falling latency with increasing intensity. Above a certain intensity



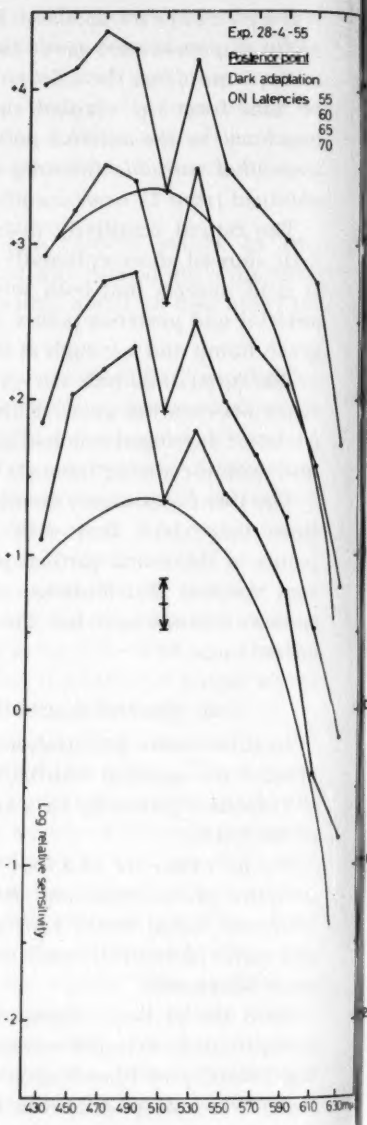


Fig. 20. See text, p. 61, and legend to Fig. 11.

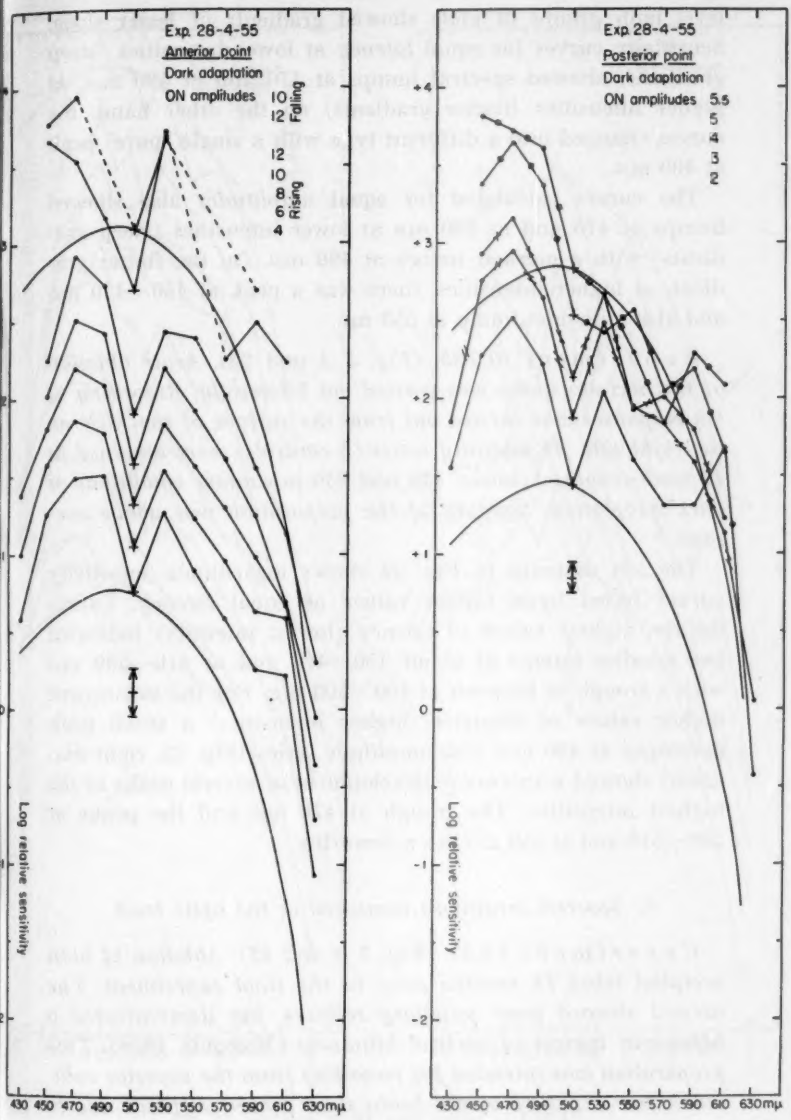


Fig. 21. See text, p. 61, and legend to Fig. 11.

level both groups of plots showed gradients of lesser slope. Sensitivity curves for equal *latency* at lower intensities (steep gradients) showed spectral humps at 470 and at 530 mμ. At higher intensities (flatter gradients) on the other hand, the curves changed into a different type with a single "pure" peak at 490 mμ.

The curves calculated for equal *amplitudes* also showed humps at 470 and at 530 mμ at lower intensities (steep gradients) with a marked trough at 490 mμ. On the flatter gradient, at higher intensities, there was a peak at 450—470 mμ and also a distinct hump at 550 mμ.

Experiment 10.6.55. (Fig. 3 A and 22). Acute ablation of the occipital poles was carried out bilaterally. Recording of On responses was carried out from the surface of the LGN on the right side. 21 intensity series (5 controls) were obtained in 17 wave-lengths between 430 and 630 mμ under conditions of dark adaptation. Stability of the preparation was above average.

The left diagram in Fig. 22 shows logarithmic sensitivity curves based upon falling values of equal *latency*. Curves for the highest values of latency (lowest intensity) indicated two smaller humps at about 450—470 and at 510—530 mμ with a trough in between at 490—500 mμ. For the subsequent higher values of latencies (higher intensities) a small peak developed at 490 mμ. The *amplitude* series (Fig. 22, right diagram) showed a successive development of several peaks at the highest intensities. The trough at 470 mμ and the peaks at 500—510 and at 550 mμ are noteworthy.

6. *Spectral sensitivity measured at the optic tract*

*Experiment 1.6.55. (Fig. 3 B and 23). Ablation of both occipital lobes 14 months prior to the final experiment. The animal showed good pupillary reflexes, but demonstrated a behaviour typical of cortical blindness (*MARQUIS *1934). This preparation was intended for recording from the superior colliculus, but exposure of the brain stem proved difficult due to scarring. Recording was made from a point on the surface of*

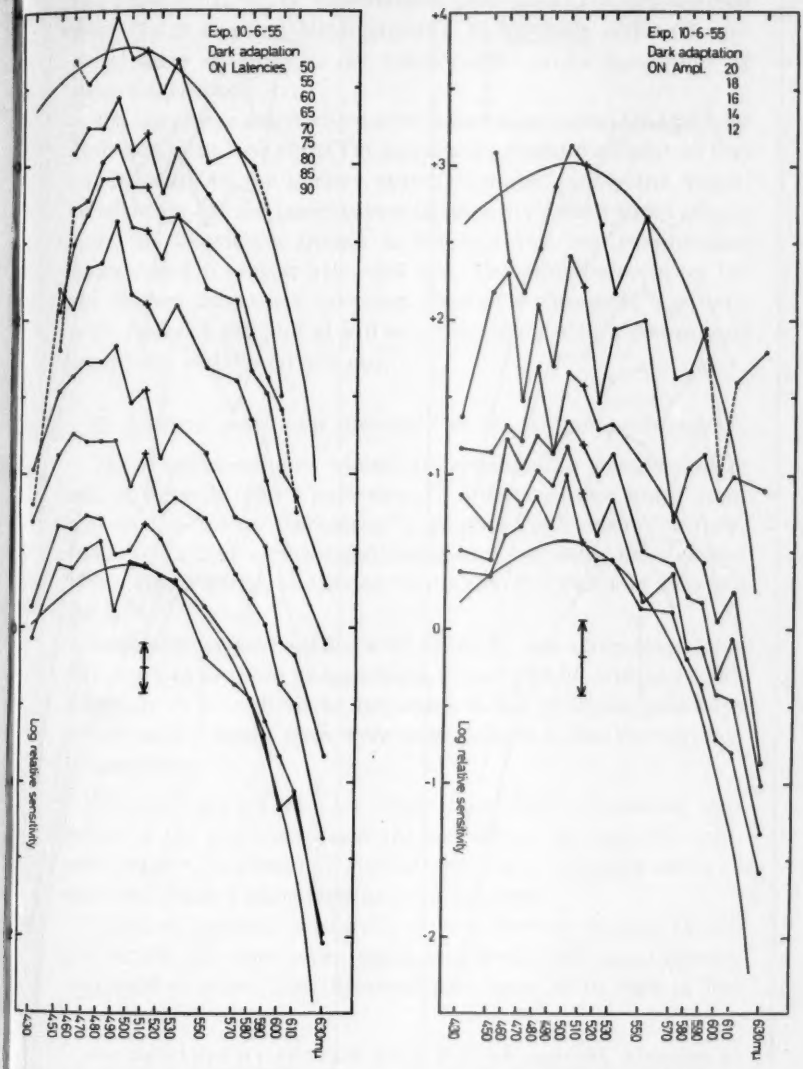


Fig. 22. See text, p. 64, and legend to Fig. 11.

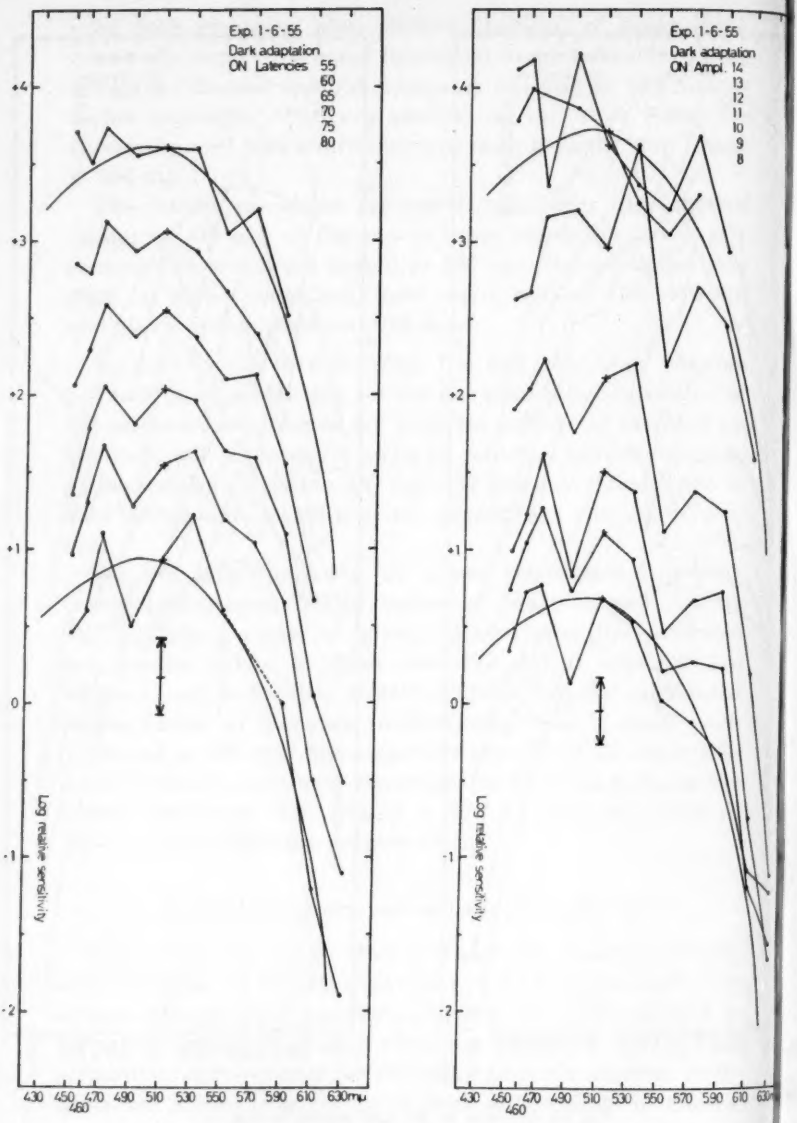


Fig. 23. See text, p. 67, and legend to Fig. 11.

the optic tract, at the approximate position of the degenerated LGN (later checked histologically). 15 intensity series (3 controls) were obtained in 12 wave-lengths under conditions of dark adaptation.

Fig. 23 shows sensitivity curves based upon equal On *latencies* (left diagram) and equal On *amplitudes* (right diagram) in this experiment. All the latency curves resemble highly the amplitude curves for the lower values of intensity (small equal amplitude) in showing a trough at 490 mμ and two surrounding humps at 470 and at 510—530 mμ. The amplitude curves for the highest intensities, however, showed a change to a pattern with peaks at 490 and at 570 mμ, coinciding with a decrease of sensitivity at 470 and 530 mμ.

7. *Spectral sensitivity measured at the superior colliculus*

These measurements aimed at investigating possible differences between the ("paleovisual") inflow to the brain stem and to the cortex ("neovisual"). A pure "paleovisual" inflow, freed from fast corticofugal backflow, can only be obtained if the visual cortex is ablated bilaterally (INGVAR and HUNTER 1955).

Collicular measurements were made in one acute (exp. 18.5. 55) and two chronic preparations (exp. 12.12.55 and 14.12.55). Attempts to record colour responses in the reticular pathways of the medial brain stem were unsuccessful in the two chronic preparations.

Experiment 18.5.55. (Not illustrated.) Recording was made of On responses from the surface of the superior colliculus under conditions of dark adaptation. 9 intensity series (2 controls) from 8 wave-lengths were obtained.

Series of spectral sensitivity curves showed humps at 470 and at 530 mμ both when equal amplitude, and equal latency was used as index. The threshold was found to be high in this experiment.

Experiment 12.12.55. (Fig. 3 C, 24 and 25). Ablation of both occipital lobes was made 19 months prior to the final experiment. During this period the animal showed good pu-

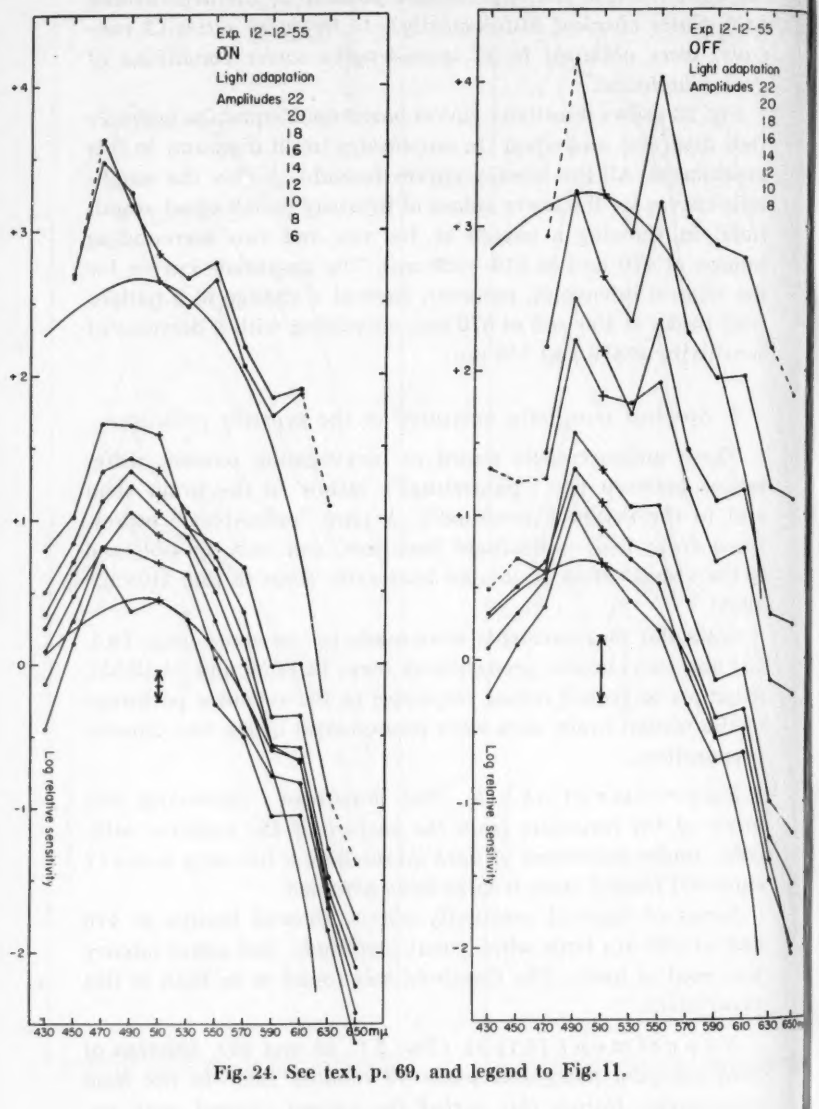


Fig. 24. See text, p. 69, and legend to Fig. 11.

pillary reflexes and signs of cortical blindness (cf. exp. 1.6.55, p. 64). Exposure of the right superior colliculus was successful and simultaneous recording of On and Off responses (flicker rate in this experiment 5.1 per second) was carried out by a lead placed centrally on the right collicular surface. Fourteen intensity series (3 controls) were obtained from 12 wave-lengths after initial dark adaptation. Subsequently, 14 series (3 controls) from 12 wave-lengths were recorded with the adapting light on after one hour of modest additional light adaptation to white light.

In Fig. 24 two diagrams are shown of sensitivity curves calculated from equal On and Off amplitudes under modest light adaptation. The On series (left diagram) showed initially, at the smallest values of equal amplitude, a trough at 490 mμ with surrounding humps at 470 and at 510—530 mμ. As the intensity increased, these humps disappeared and a maximum developed at 490 mμ which broadened at higher intensities. The sensitivity curves for the highest values of equal amplitude showed peaks in the blue (470 mμ), in the green (550 mμ), and also a small hump at 610 mμ.

In the Off series (right diagram) the smallest amplitudes indicated a sensitivity with a clear maximum at 490 mμ. This was retained with increasing intensity and the final curves showed a second large peak at 550 mμ. The intensity of the spectrum did not suffice for a complete exploration of the blue part. All the On and the Off curves in Fig. 24 demonstrate a small hump at 610 mμ. This peak is discussed on p. 86.

Experiment 14.12.55. (Not illustrated.) Ablation of both occipital lobes was made 19 months prior to the final experiment. The animal showed good pupillary reflexes and signs of cortical blindness. Recording of On responses was carried out from the right collicular surface with a constant flicker rate of 5.1 per second. Fourteen intensity series (3 controls) were obtained from 12 wave-lengths after the initial dark adaptation. Some adjustments of electrode positions, light beam etc., then became necessary so that the ensuing 14 series (3 controls) from twelve wave-lengths in modest additional light adaptation can-

not be compared with those obtained under conditions of dark adaptation. Instability made this experiment less satisfactory.

Calculations for the first series in dark adaptation gave curves (for equal latency and amplitude) with deviations from the rhodopsin distribution at 490 mμ and, at other levels of intensity, a small trough at 490 mμ and surrounding humps at 470 and at 530 mμ. In light adaptation the threshold was found to be high and a smaller deviation developed at 590—610 mμ.

B. ANALYSIS OF DEVIATIONS FROM THE RHODOPSIN DISTRIBUTION OF SPECTRAL SENSITIVITY

It is evident from the logarithmic diagrams presented above that deviations from the rhodopsin distribution of spectral sensitivity expressed by the cerebral responses were very numerous, and that gradual shifts from one pattern to another were often recorded. As expected, the deviations were much larger with higher stimulus intensities, or against a background of modest additional light adaptation. Primarily, it would therefore seem justified to differentiate between these large high-intensity deviations and those calculated from small responses of long latency at low intensities.

1. *Deviations in light adaptation*

The deviations obtained with weak additional light adaptation closely resembled those obtained under conditions of initial dark adaptation but with the highest intensity of flicker. These will therefore be dealt with together.

It was a regular finding that light adaptation produced a spectral peak at 550 mμ. Since it seems reasonable to assume that this peak is related to the "photopic dominator" of GRANIT (1945 *et seq.*) the spectral sensitivity distribution encountered together with a well formed peak at 550 mμ may be defined as the *photopic pattern* and thus ascribed to cone activity (*cf.* Fig. 30). Examples of this pattern are found in the logarithmic diagrams of Fig. 14 (right diagram), 16 (left) and 24. As

seen in these figures, the peak at 550 mμ was regularly combined with a markedly raised sensitivity in the blue (430—470 mμ). In percentual curves the sensitivity in the blue and green often reached several hundred per cent (*cf.* Fig. 28 and 29). There was also very often with higher intensities a small hump around 610 mμ (Fig. 20 and 24).

2. *Deviations at threshold and at lower intensities*

It will be recalled that the "threshold" for the cerebral responses was defined as the lowest intensity step at which responses could be traced in the photographic records. Thus, the distance in terms of intensity from the absolute threshold to "cortical threshold" here is unknown. As described, however, stimulation with flicker was carried out in each wave-length starting with intensities 0.5 to 1 log unit below cortical threshold (*cf.* Fig. 6) after initial dark adaptation for at least one hour. At such low intensities a fully dark adapted human observer could not distinguish colours when looking into the monochromatic light beam. It is thus likely that the sensitivity distributions obtained from small responses of long latency with low intensity of flicker were recorded in what is commonly defined as the scotopic state (*v. i.*, Discussion p. 83).

It was an unexpected finding that most of the curves calculated from equal responses with small amplitude or long latency (low-intensity responses) often showed greater deviations from the rhodopsin curves than was found at certain other intensity levels. However, in the study of several hundred sensitivity curves it was found that certain deviations occurred more regularly than others. Series of sensitivity curves for increasing intensity furthermore suggested that gradual systematic shifts of certain spectral peaks around the reference point at 510 mμ occurred. It was thus possible to differentiate two main patterns at low intensity which have been termed *scotopic patterns* (*cf.* Fig. 30), since none of them showed a peak at 550 mμ. These two low-intensity patterns are probably related to rod reception.

One of the main low-intensity spectral sensitivity patterns had its maximum at 490 mμ. This form of spectral curve was,

in fact, the only one encountered in a "pure" single peaked form (*e. g.* Fig. 25). The remainder of the curves usually showed several peaks. When a peak at 490 mμ was present and was followed for several steps of intensity, it was a rule that the sensitivity values at 450—470 mμ and at 530 mμ decreased, if the 490 mμ value was increasing. On the contrary, if the 490 mμ value decreased the two peaks at 470 mμ and — with somewhat less regularity — at 510—530 mμ developed. The pattern containing two main peaks at 470 and 530 mμ bordering a trough at 490 mμ has been interpreted as the other main scotopic pattern. There was thus a definite suggestion in the records of a "rivalry" between the two scotopic patterns. Examples of gradual changes between the two scotopic patterns will now be given.

Fig. 25 gives a series of percentual diagrams from exp. 12.12.55 (collicular recording under weak additional light adaptation) in which percentual sensitivity curves were calculated for narrow steps of increasing equal On amplitude (*cf.* Fig. 24). Each of the small curves is based upon a scale in which every twentieth mμ has been marked (430—630 mμ). The perpendicular white line indicates the "average" reference point (*cf.* p. 32), which is the 100 per cent value at 510 mμ. The numbers indicate equal amplitude value in arbitrary units for the respective curves. Curves from 5.5 to 8.0 show a maximum at 470 mμ and a hump at 510—530 mμ. However, with increasing amplitude the 470 mμ value falls and simultaneously the 490 mμ value increases so that from 8.5 and up to 17.5 it constitutes the maximum. The peak at 490 mμ describes a gradual increase with increasing intensity and reaches a peak in the "pure" curves (*v. s.*) calculated for equal amplitudes around 13.0. It then gradually falls and, finally, at the highest intensities, a third pattern — the photopic — develops with a peak at 470 mμ and suggested humps at 550 and at 610 mμ.

In Fig. 26 from exp. 13.6.55 a similar sequence of changes is pictured. These diagrams pertain to cortical Off responses of equal amplitude recorded under conditions of dark adaptation (*cf.*, Fig. 12). The diagrams of the first row all show two peaks at 450—470 mμ and at 530 mμ. However, these peaks diminish

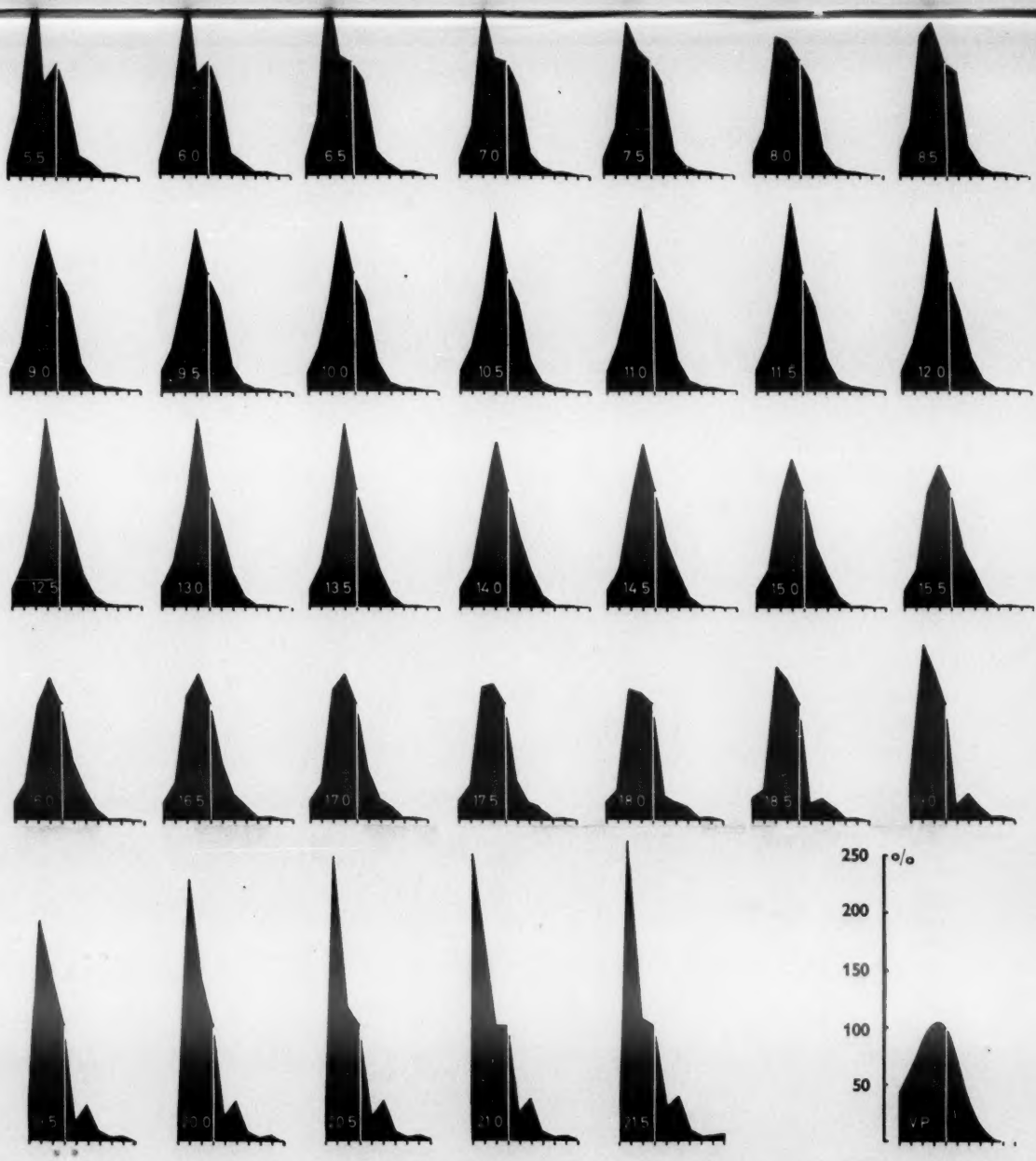


Fig. 25. ***Exp. 12.12.55.*** **Percentual spectral sensitivity curves calculated for narrow steps of increasing equal amplitude (indicated on each curve in arbitrary units). Every small curve is based upon a dotted line on which the dots indicate every 20th mμ from 430 to 650 mμ. The 100 % reference value at 510 mμ corresponds to upper end of the white perpendicular bar in each small curve. The curves were calculated for "average" reference values (see text, p. 32). Diagram in lower right corner indicates rhodopsin absorption.**

There is a gradual change of spectral sensitivity from the patterns experessed by small responses at low intensities to the patterns expressed by larger responses at higher intensities of flicker. Note change of initial maximum at 470 mμ (amplitudes 5.5 to 8.0) to maximum at 490 mμ (amplitudes 8.5 to 17.5). The final high-intensity pattern shows a large peak at 470 mμ and a definite submaximum at 550 mμ. There is also an indication of a hump at 610 mμ. See text, p. 72.

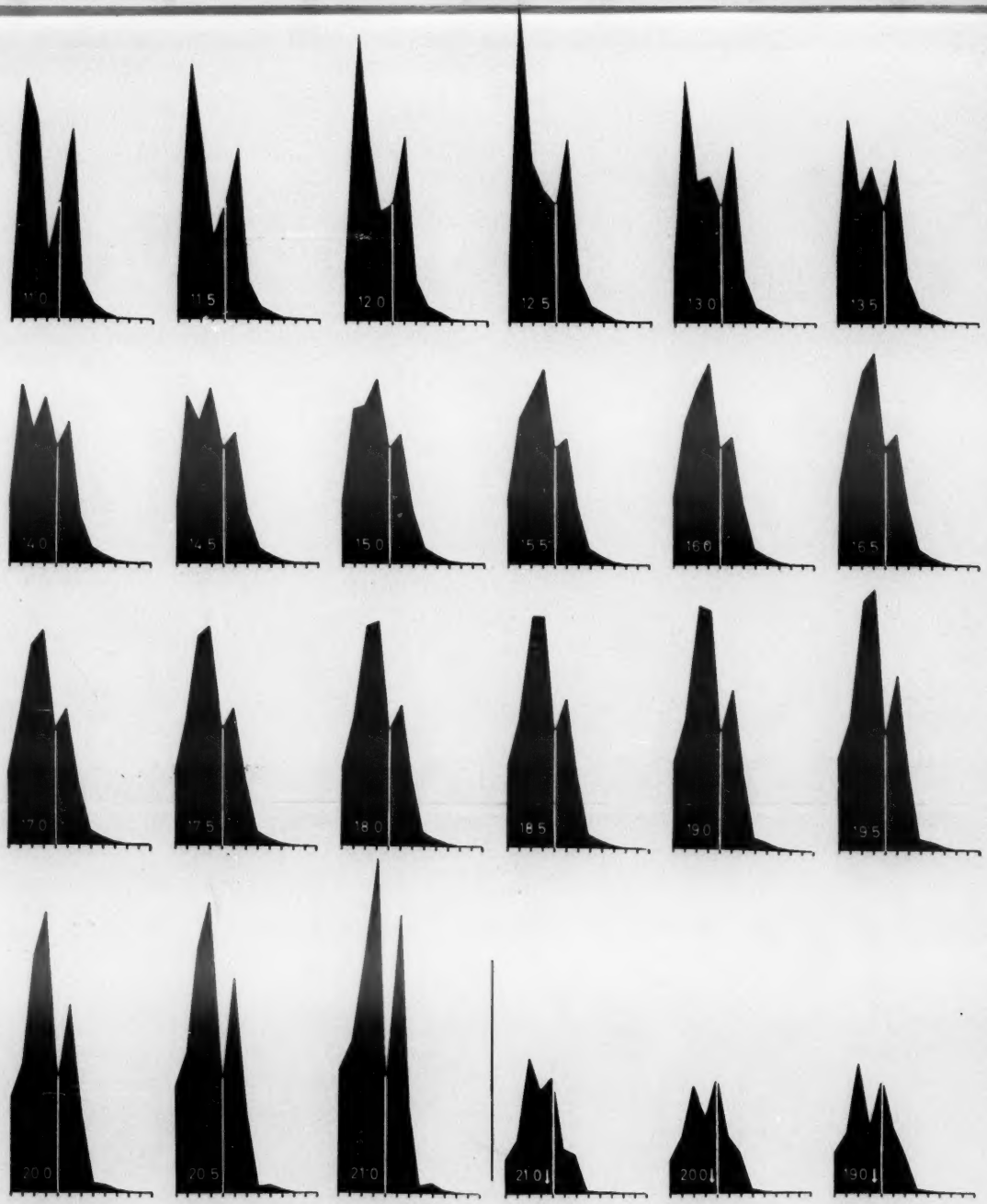


Fig. 26. ***Exp. 13.6.55.*** **Sensitivity curves for narrow steps of equal amplitude of cortical Off responses. Last three curves calculated from "falling phase" of amplitude (see text, p. 48).**

See also legend to Fig. 25 and text, p. 72.

when the value at 490 mμ increases to become the maximum at amplitudes 14.5 to 21.0. The final three curves of Fig. 26 were calculated from the falling gradients of amplitude at the higher intensities. It is readily seen that in the falling phase the very large maximum at 490 mμ has disappeared and, instead, there is a trough at this spectral position with two surrounding peaks at 470 and at 530 mμ — a pattern similar in principle to the initial one for equal amplitude 11.0 at low intensities. Similar shifts between the two low-intensity patterns were regularly found in the dark adapted state when the spectral sensitivity was followed from the rising to the falling phase of amplitude in the original intensity plots.

3. *Controls*

Due to the variations in the preparations, as well as in the measurements (*v. s.*, p. 31, and Discussion, p. 81), the changes in spectral pattern were further investigated in all experiments by the calculation of percentual sensitivity values both in relation to an "average control value" and to a "running control" (*v. s.*, p. 32). If these calculations demonstrated that the difference between the "average curve" and the "running control curve" was large, it implied that the experimental conditions had been unstable, the records difficult to measure, or that the mechanisms investigated had been unstable. As mentioned, several sensitivity curves were discarded due to the fact that instability had been present.

However, the main high-intensity and the two low-intensity patterns described above were encountered in a sufficient number of experiments in which the respective peaks and troughs of the sensitivity curves lay outside variations caused by "instability" (*v. i.*, p. 81). This fact, together with the systematic shift between the patterns with increasing intensity suggested that these spectral sensitivity patterns were caused by characteristics pertaining to the receptor systems studied.

One further fact supporting this suggestion was that the two main low-intensity patterns in some experiments were recorded differentially in the On and Off component of the same cerebral response complex. Fig. 27 demonstrates such an

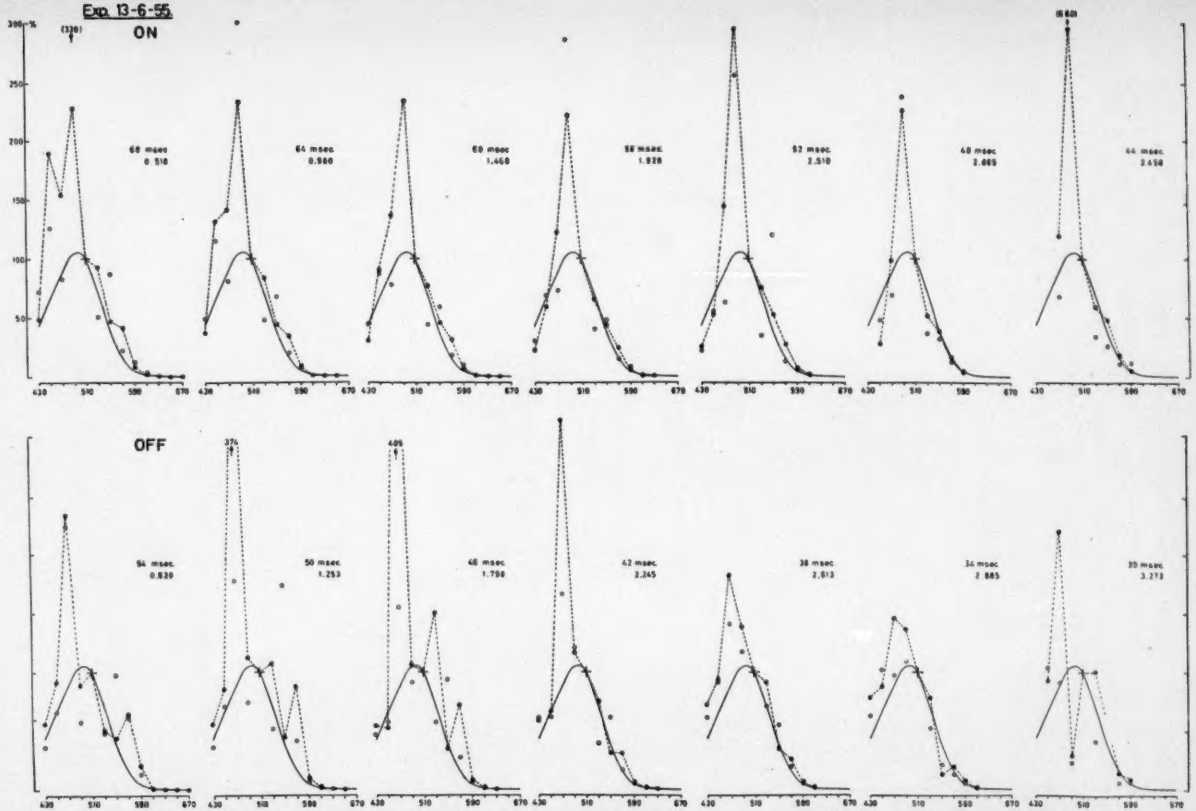


Fig. 27. *Exp. 13.6.55.* Percentual spectral sensitivity curves calculated for steps of equal latency of cortical On (upper row) and Off (lower row) responses. Black dots (or figures) joined by dashed lines indicate sensitivity in relation to "average controls" (intensity level indicated in log units below equal latency figure). Circles (or figures in parenthesis) give sensitivity values in

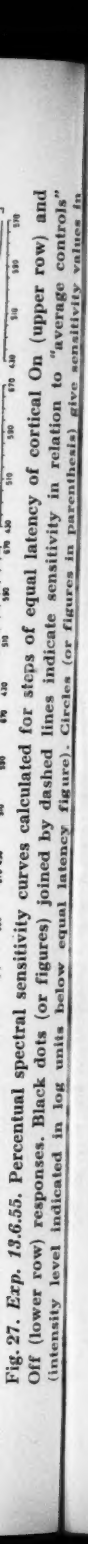


Fig. 27. *Exp. 13.6.55.* Percentual spectral sensitivity curves calculated for steps of equal latency of cortical On (upper row) and Off (lower row) responses. Black dots (or figures) joined by dashed lines indicate sensitivity in relation to "average controls" (intensity level indicated in log units below equal latency figure). Circles (or figures in parenthesis) give sensitivity values in

example from exp. 13.6.55 (*cf.* Fig. 11). The On series (upper row) shows a very large and relatively "pure" maximum at 490 mμ. It is readily seen that this peak is determined by values which lie well outside the variability as expressed by the distance between dots and circles. The Off series (lower row), on the other hand, shows a large initial peak at 470 mμ and significantly low values at 490 mμ. When the peak at 470 mμ decreased (equal latency 46, 42, 38 and 34 msec) it is seen that the values at 490 mμ increased. On the whole, the variability was greater in this example in the Off series than in the On curves since the distances between dots and circles are larger. This was, as mentioned, a frequent finding for Off responses in dark adaptation. Large distances between dots and circles were also found when, as in this case, the index measured expressed a shifting pattern. This has been interpreted to show a greater inherent instability of the mechanisms represented in the Off component.

In this series of Off curves only the peak at 470 mμ (and perhaps at 570 mμ) seems significant, while there is great instability in the 530—550 mμ region. The general type of the Off series nevertheless justifies its classification as the pattern which often appeared as "rivalling" the one with a peak at 490 mμ in the sense that many experiments showed this shift between a spectral hump at 490 mμ and another pattern with two surrounding peaks at 470 and 530 mμ.

In other experiments the "rivalry" was not so evident. There was then a general expansion of sensitivity in the blue-green with less specification of narrow-tipped maxima. At present, there is no explanation why the two low-intensity patterns sometimes appeared mutually exclusive, sometimes additive, to deliver sensitivity values far above the rhodopsin distribution of sensitivity.

Two further examples of the variability of the spectral curves are given in Fig. 28 and 29 from exp. 3.6.55 in which light adaptation was used during cortical recording (*cf.* Fig. 14 and Fig. 16). The On series in dark adaptation (Fig. 28, upper two rows) shows a probable maximum at 490 mμ for the rising phase of amplitude, while on changing the gradient a large peak

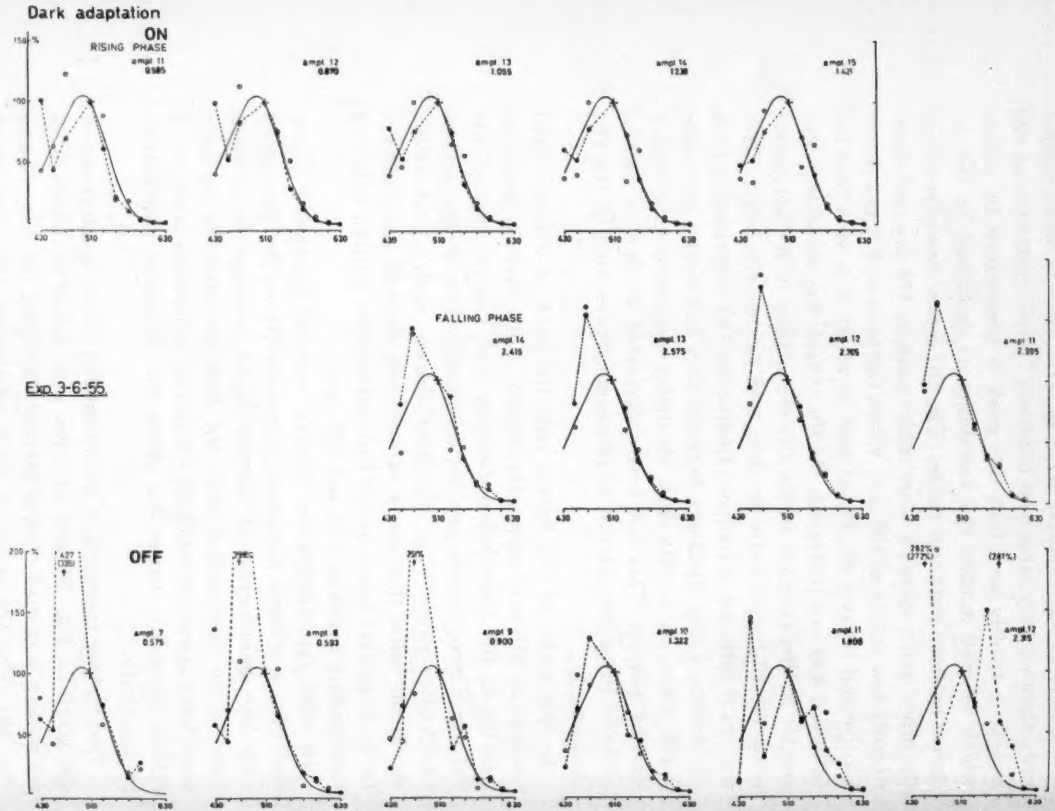


Fig. 28. *Exp. 3.6.55.* Conditions of dark adaptation. Percentual sensitivity curves for On and Off responses of

Fig. 28. *Exp. 3.6.55.* Conditions of dark adaptation. Percentual sensitivity curves for On and Off responses of

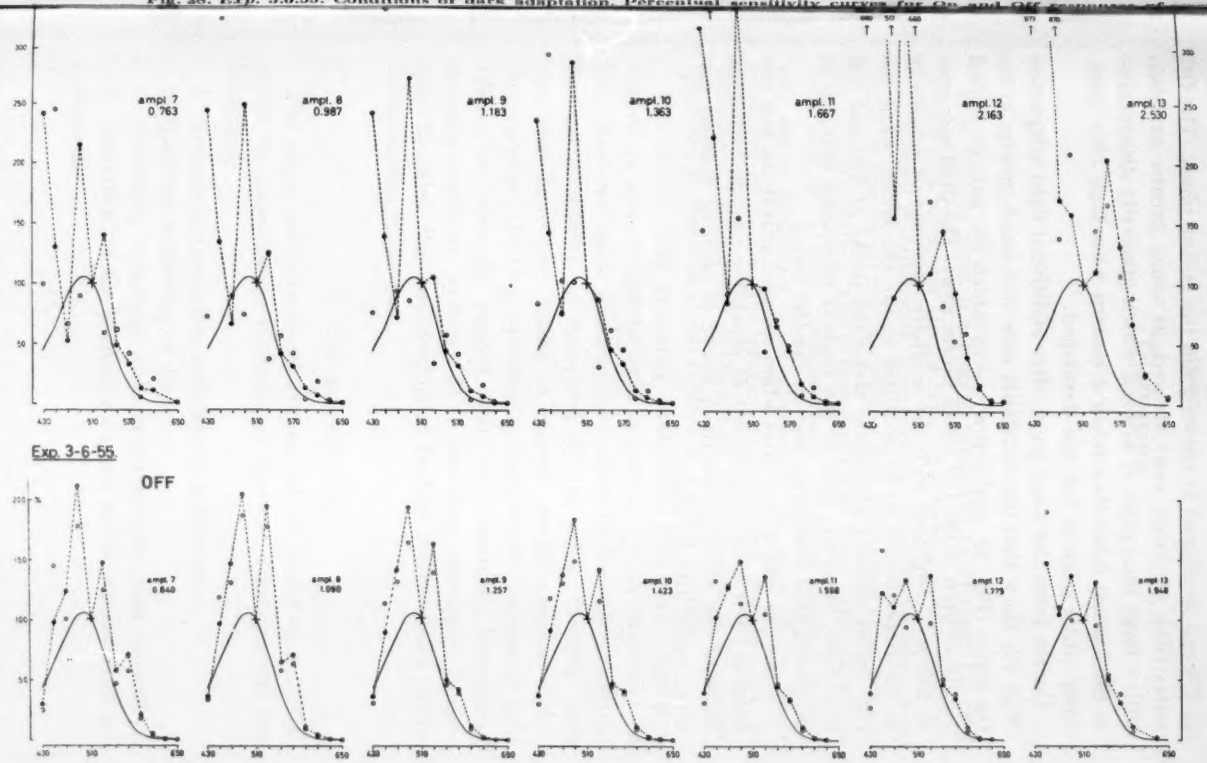


Fig. 29. *Exp. 3.6.55.* Conditions of light adaptation. Symbols as in Fig. 27. See also Fig. 16 and text, p. 78.

at 470 mμ developed in curves from the falling phase. The Off series (Fig. 28, lower row) showed, as usual, greater variation with a large blue peak at 470 mμ which gradually disappeared at the highest intensities when a pattern with high blue sensitivity and a peak at 550 mμ developed.

Curves from the same point after additional light adaptation (Fig. 29) show that the instability now was much greater in the On series (Fig. 29, upper row) — in which the pattern shifted highly — than in the Off series (lower row). The photopic peak at 550 mμ combined with the dramatic increase of sensitivity in the blue seemed to be confined to the On series, while the Off curves showed ultimately stabilized peaks at 490 and 530 mμ. Note, however, that at the highest intensities there is an increased blue sensitivity also in the Off series.

It may be added here that when spectral sensitivity was calculated from equal amplitudes of *repetitive* cortical responses, some of the features described above of shifting spectral patterns could also be seen.

Chapter IV

Discussion

It is evident from the results presented in chapter III that instead of a relatively simple relation between retinal and cerebral spectral distributions, unexpectedly complex patterns were found in the visual centres even at low intensities after preceding dark adaptation. The demonstration of these patterns, which show new, or, at least, unconventional forms of spectral curves, sets many problems of interpretation. In spite of its fairly extensive character, the present investigation should only be considered a pilot study of the neural transmission of the visible spectrum to cerebral visual centres in a single species.

The results have primarily established that mass responses evoked by monochromatic light in cerebral visual structures do not follow one single function determined by the average retinal spectral sensitivity as measured by the ERG. Instead, such responses have been found to express certain spectral distributions which, by their systematic changes with increasing light adaptation, strongly suggest that they represent interacting receptor-conductor systems. It is therefore necessary to consider in detail the methodological factors which could throw doubt on this suggestion.

1. *The method used*

The main methodological factors which could have influenced the present determinations of spectral sensitivity are the following:

A. Preretinal absorption and tapetal reflection.

B. Entoptic scattering of light.

C. Recording technique and measurements from records.

D. "Stability" of the preparations and of the mechanisms investigated.

A. In chapter II the reasons have been given for not using a correction for *preretinal absorption* and for *tapetal reflection* (*v. s.*, p. 14). As to the most important of these two factors, tapetal reflection, the following may be added. The recordings made from the brain, especially when two cortical points were studied simultaneously (p. 60), probably represented both intra- and extratapetal retinal loci. The mass responses from the surface of the LGN or the colliculus may have represented both simultaneously. Since the main patterns were encountered in all experiments, no matter from where recording was made, this would exclude any significant contribution of tapetal reflection to the differentiation observed. The spectral variation in tapetal reflection is also much too low to explain the large peaks found in the different patterns. The systematic variation of the patterns with increasing intensity, and especially, the differential simultaneous recording in On and Off components of the two different low-intensity patterns (p. 49) would, it seems completely rule out a greater rôle played by tapetal factors. Admittedly, however, such reflection could augment individual values in the curves. With GRANIT (1943) and GUNTER (1952) one is inclined to interpret the small irregular hump at 570 mμ (often occuring at low intensities together with the peaks at 470 and 530 mμ; Fig. 29) as due to the tapetum which has its maximal reflective capacity in this spectral location (WEALE 1953 a and d).

B. Recently DEMOTT and BOYNTON (1958 a and b) have analyzed the sources and distribution of entoptic *stray light* in the bovine eye. The cornea was found to be responsible for 75 per cent of the scattering and the lens for the rest. Careful measurements revealed only smaller differences in light scatter between different wave-lengths. Previously BOYNTON (1953) had shown that the effects of stray light upon the ERG are very small provided a large area of illumination is used. In view of these findings, and since diffuse illumination of the eye with widely dilated pupil was used in the present experiments, it seems safe to conclude that the effects of stray light upon the spectral sensitivity determinations have been small. With GRANIT and WIRTH (1953; *cf.* JOHNSON and CORNSWEET 1954) it

is also concluded that the blue shift of retinal spectral sensitivity obtained by the ERG flicker technique cannot have been caused by stray light effects.

Since thermal energy may bleach rhodopsin (St. GEORGE 1952), a filter for long wave-lengths (p. 26) was used in the main experimental series. Earlier experiments carried out without this filter also showed the presence of the three main spectral patterns reported above.

C. By the use of small input capacitances (p. 28) a substantial amount of the ERG background, as well as slower response components, was excluded by the *recording technique.* Controls with larger condensors demonstrated that no certain difference in "cortical threshold" (*v. s.*, p. 27) was obtained with the time constants used. However, the derivation of the responses with the short time constant might have accentuated some of the rising and falling phases of amplitude such as those shown in Fig. 8 and 10.

The error caused by measurements of the records (± 3 % for latency measurements and ± 10 % for amplitudes) must be considered relatively small in comparison to the variability caused by changes in the preparation and by the inherent instability of the mechanisms investigated (*v. i., D.*).

D. The *stability of the preparations and of the mechanisms investigated* was, as mentioned, judged by the variations found in the control series at 510 mμ. Several experiments were made with a uniform technique and simplified graphical methods (p. 31) were used to indicate the variations in the diagrams. 'Stability' was considered satisfactory when the controls did not vary more than ± 0.10 log. units (about ± 12.5 %) during 3—4 hours. Variations of this order were generally only found in measurements of latency of On components of the responses (Fig. 13 and 15). The mean variation for 7 experiments in which 27 series of 177 sensitivity curves were calculated was, however, higher, ± 0.20 log units (0.17—0.62), which is about ± 16 %.

It is evident that the large changes of sensitivity obtained with light adaptation, or with the highest intensities of illumination, changes termed photopic, are fully significant (Fig.

15, 16, 24 and 25). On the other hand, many of the humps and troughs in the spectral curves at lower intensities will — *in the individual sensitivity curves* — represent random variation around a rhodopsin distribution. However, as pointed out previously, several common trends and systematic changes were repeatedly found *in all of the series of sensitivity curves.* These changes support the general conclusion that, depending upon the level of intensity used, systematic shifts of pattern do occur leading to or from (large) significant peaks across intermediate patterns in which it is impossible to deduce much from individual stages. Some of the peaks and troughs, even at the lowest intensities, lay well outside the variation factor pertaining to the experiment.

2. *Origin of spectral patterns found in cerebral visual centres*

Evidence has been presented above for the view that the main cerebral spectral patterns recorded above are characteristic of the optic system of the cat. The retinal origin of the patterns is proved by their presence in the measurements from the optic tract (exp. 1.6.55, p. 64) after removal of the visual cortex and complete degeneration of the principal cells of the LGN.

The retinal origin of the cerebral spectral patterns was, in fact, already signalled by the deviations from the rhodopsin curve found in all experiments in the determinations of average retinal spectral sensitivity (p. 34—38). In view of the results obtained in the visual centres, it seems likely that the "blue-shift" of retinal sensitivity obtained with flicker (GRANIT and WIRTH 1953) represents an average shift of sensitivity which is determined by at least two interacting scotopic receptor mechanisms displaying a high sensitivity in the 470—490 mμ-region, one of which must be more blue-sensitive than the other.

The anatomy of the cerebral visual pathways (p. 9—14) suggests that a gross lead placed upon the visual cortex would register an activity representing a limited retinal population of receptors. Since the distribution of rod and cone receptors over

the retinal surface is not uniform one could expect that two points recorded from simultaneously on diffuse retinal illumination would not express identical spectral sensitivity. In exp. 16.5.55 and 28.4.55 (p. 60—61) in which this deduction was tested, different spectral patterns were found in the two points at certain intensity levels. Such differences are most likely explained by the retinal anatomy.

3. *Light adaptation in flicker*

The rich and systematic variation of spectral sensitivity patterns shown above would seem to agree with expectations from current physiological and anatomical work (*v. s.*, p. 9—14, and 15—19). Even at low intensities, in the scotopic range. when rod activity should dominate at all visual stations, there are clearly two main varieties of pattern. One of these has a high sensitivity at about 450—470 mμ and it apparently interferes, sometimes predictably, sometimes unpredictably, with what may be presumed to be the rhodopsin pattern which then emerges as a sharp peak at 490 mμ.

By the flicker method a certain amount of light adaptation is always brought into play. Strictly speaking, therefore, this method cannot be applied to measure spectral sensitivity in the fully dark adapted state which ends as soon as the retina is illuminated and does not return until after a considerable period of total darkness (BARLOW, FITZHUGH and KUFFLER 1957). However, according to the definition given above (p. 70) only those patterns which contained a definite spectral peak at 550 mμ have been identified as *high-intensity ("photopic") patterns* suggestive of cone activity which is well known to possess high sensitivity in this region. Patterns recorded at lower intensity levels have been tentatively ascribed to rods.

4. *The two low-intensity patterns*

It seems reasonable to assume that one of the low-intensity patterns with its sharp single peak at 490 mμ is determined by

dominant light absorption by rhodopsin. This pattern was mostly found to dominate in the scotopic state, *i. e.* a peak at 490 mμ was present in the majority of curves in which no trace of the photopic pattern was seen. Independence of the 490 mμ pattern (from the one with peaks at 450—470 mμ and at 510—530 mμ was, as mentioned, clearly evident by differential simultaneous recording in On and Off response components, and also by the phenomenon called "rivalry" (*v. s.*, p. 72) when it was seen. There was, however, no clearcut sequence in the rivalry, *i. e.* with increasing intensity (light adaptation the 490-pattern would be followed after depression by the other pattern or the opposite. Thus, both low-intensity patterns co-existed in the scotopic state in a fluctuating manner so that sometimes the 490-pattern, sometimes the other was dominant in the index measured. It is further of interest that when the photopic pattern developed gradually — this was as a rule most clearly seen in the On component — a large peak at 490 mμ often persisted in the Off curves together with some of the characteristics of the photopic pattern (Fig. 16 and 21). In accordance with the general interpretation above of the low- and high-intensity patterns, this suggests the persistence of some rod function in the photopic state when the cone activity is dominating.

As mentioned, it seems natural to conclude that the 450—470 mμ peak, seen at low intensity, are expressions of mechanisms which, in part, cause the retinal blue-shift demonstrated in ERG mass responses by GRANIT and WIRTH (1953) and confirmed above.

These authors suggested that "fresh labile blue-sensitive systems" (iso-rhodopsine, DARTNALL's pigment 467) created by bleaching of rhodopsin could be causing the shift. Recent measurements by WULFF *et al.* (1957) and by ADAMS *et al.* (1957) of rapid absorption changes in rhodopsin after illumination with a flash would support this assumption. It may also be recalled here that the flicker method in man, when used at low levels of adaptation, has also sometimes yielded blue-shifts of spectral sensitivity. It seems likely that the "inverse Purkinje shift" recorded by IVES (1912) and by SLOAN (1928; *cf.* DE VRIES 1948) with flicker photometry represent phenomena related to the findings here.

The low-intensity peak at 510—530 mμ in many instances behaved independently of the other main peak at 450—470 mμ, being variable in size and sometimes persisting at higher intensities than the blue peak (Fig. 26). Spectral humps at 520—530 mμ have been recorded by GRANIT (1941, 1945) after selective bleaching or polarization and they were held to indicate "green modulator" functions.

5. *The high-intensity pattern*

The blue peak (450—470 mμ) was the largest one of the high-intensity pattern. On increasing stimulus intensity it never developed directly out of the blue peak of the low-intensity curves which usually had its peak at 470 mμ. There was generally an intermediate stage in which the 490-peak dominated (Fig. 25 and 29).

In some experiments, a very high sensitivity was recorded in the shortest wave-lengths tested (430—450 mμ). This was found with high intensities, or after additional light adaptation (Fig. 15, 16, 19 and 23). Such curves indicate the presence of a further blue-sensitive mechanism "outside" (at 430 mμ or lower) the well defined peak at 450—470 mμ. It seems a likely possibility that such "far blue" high sensitivity might represent absorption by some final bleaching product of rhodopsin such as beta-rhodopsin.

The green peak at 550 mμ has, as mentioned, been ascribed to the photopic dominator of GRANIT (1945, 1955 a). This peak usually only attained one fifth of the blue and was often broad (Fig. 16, 19, 24 and 25).

WALD, BROWN and SMITH (1955) have pointed out that the photopic dominator of several species (frog, snake, cat) shows a spectral curve which resembles the absorption spectrum of iodopsin. They have also suggested that iodopsin might represent the middle member of the three different photopic mechanisms responsible for colour vision in man which have been identified by psychophysical methods by STILES (1939 *et. seq.*). This suggestion seems compatible with the results obtained here in which a pure photopic dominator curve with a single maximum at 550 mμ was never recorded, in spite of additional light adaptation. In the photopic state at least two main spectral peaks and a suggestion of a third hump, were always seen.

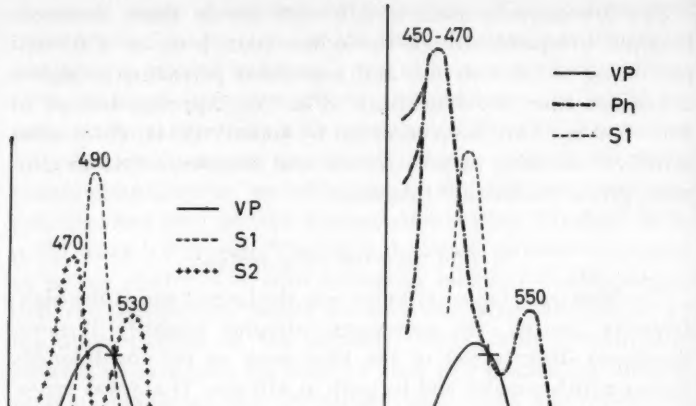


Fig. 30. Schematic diagram summarizing the spectral patterns obtained from responses to monochromatic flicker in cerebral visual centres. The *left* diagram summarizes the two main *low-intensity* patterns (called S 1 and S 2 here) and indicates their main spectral peaks in relation to the rhodopsin absorption curve (VP). The *right* diagram shows the main *high-intensity* pattern (called Ph) and indicates its relation to the rhodopsin curve (VP). The persistence of a peak at 490 mμ (probably S 1) at high intensities has also been indicated as well as the very high "far blue" sensitivity (at 430 mμ or lower) which was sometimes seen in the light adapted state.

In the red, at about 590—630 mμ (usually at 610 mμ), a stable deviation was often seen, even at low intensities (Fig. 12, 14, 20, 21 and 23), and also together with the photopic peaks in the blue and the green (Fig. 24 and 25). It is interesting to speculate whether this small peak is related to presence of small amounts of the erythrolabe pigment identified by RUSHTON (1957) in man and frog by his method for density measurements of retinal pigments *in vivo* (RUSHTON 1953 b).

In Fig. 30 a schematic diagram is shown in which the three main spectral patterns found in the cerebral visual centres have been outlined in relation to the rhodopsin absorption curve (V P). The two low-intensity patterns have been labelled "S 1" and "S 2" (S=scotopic) and the high-intensity pattern "Ph" (=photopic). The diagram shows only hypothetical "pure" patterns. The fluctuating interaction between them and transitional states have been disregarded.

6. *Retinal mechanisms underlying the patterns recorded in the brain*

In 1953 (d), WEALE demonstrated with a direct method that the cat's retina *in vivo* contains both slowly and rapidly regenerating visual pigments. With the technique used in the present experiments, which implies a selective adaptation during recording of each "intensity series", both slow and fast adaptation mechanisms were able to affect the cerebral responses. It is believed that several of the findings above would have been missed by a more rapid experimental procedure. A good indication for this view is provided by the experiments in which cortical responses were recorded to various wave-lengths with intensities adapted to the ERG sensitivity curve (*v. s.*, p. 38). In such experiments only smaller differences could be seen between the responses which were recorded in rapid succession.

It seems of great interest that the difference spectrum obtained by WEALE (1953 d; *cf.* DARTNALL 1957) directly in the cat's retina for the — "probably complex" — slow regenerative process has its maximum at about 500 mμ and two humps at 470 and 530 mμ respectively. This distribution coincides strikingly with some of the composite curves of the two main low-intensity patterns which were recorded in cerebral visual centres. It also resembles some of the retinal sensitivity curves (*e. g.* Fig. 2 A). The (occasional) fast process of WEALE (1953 d) had its maximum at 550 mμ.

In the studies of GRANIT and his collaborators there are several experimental findings which suggest that the patterns recorded here in the visual centres mirror the sensitivity expressed by single retinal ganglion cells under certain conditions (GRANIT 1943, 1945, 1948; *cf.* summaries 1947 and 1955 a, *cf.* p. 15—18). As pointed out by GRANIT (1955 a), "pure" modulator curves were not obtained in the cat. Procedures like selective adaptation or polarization were necessary to show selective spectral sensitivity in isolated retinal elements. The recorded curves in the cat were often complex, containing two or more humps.

In view of the importance of light adaptation in flicker (p. 83), it is of interest that DONNER and GRANIT (1949) found a scotopic sensi-

tivity for On elements which coincided with the rhodopsin distribution, while On-Off elements often showed an increased sensitivity in the blue. Some of their On-Off curves do, in fact, highly resemble the scotopic pattern described above with peaks at 470 and 530 mμ. Similar curves were also found by GRANIT (1945; *e. g.* Fig. 5, p. 201) with modest white, or selective adaptation. With light adaptation the spectral sensitivity of an element demonstrating the scotopic dominator curve could also change into one with a sharp maximum at 490 mμ.

The difference between measurements of spectral sensitivity at threshold and with flicker is emphasized by the findings of DODT (1956 b) in the rabbit in the photopic and the scotopic state. *At threshold* he found sensitivity to coincide with the scotopic and the photopic dominator curves. Taking another index, however, "excitatory" and "inhibitory" fusion points *in flicker* of increasing intensity, he found several forms of deviations from the two fundamental dominator curves. The deviations thus found in the rabbit (with coupled spectral peaks at 460 and 530 mμ) coincide strikingly with one of the main low-intensity spectral patterns found in the visual centres of the cat. These results in the rabbit — a species which lacks a tapetum — indicate that scotopic light reception is heterogenous in other species besides the cat (concerning the guinea pig, *v. i.*, p. 89). This heterogeneity would seem to require the flicker method to be demonstrable.

It is not possible at present to relate the cerebral spectral patterns to certain photopigments since, apart from rhodopsin, so little is known about these *in vivo*. The extreme sharpness of many of the cerebral spectral peaks would imply a first obstacle for a comparison with the more rounded absorption curves of pigments found in different retinae or chemically synthetized. As an alternative to narrow-banded pigments GRANIT (1945, 1955 a), DONNER and GRANIT (1949), and recently also DONNER (1957), have suggested that retinal neuronal interaction may "sharpen up" initial broad band absorption by some process mimicking alinear amplification. The work of SVAETICHIN (1956 a, b, c) of MACNICHOL, MACPHERSON and SVAETICHIN (1957) and of MOTOKAWA, OIKAWA and TASAKI (1957), in carp, and of GRÜSSER (1957) in cats, would suggest that very sharp spectral peaks are not recorded from units which probably are situated in the layer of bipolar and horizontal cells in the retina.

7. *Concluding remarks*

A comparison of the results from cortical, collicular and LGN recordings indicates principal similarities in spectral projection between these cerebral visual stations in the cat. In all three

centres two low-intensity and one high-intensity pattern were encountered. It may therefore be concluded that, in the cat, the sharply defined spectral messages, demonstrated early by GRANIT (*cf.* summary 1955 a) in single retinal ganglion cells of the retina of eyes with a larger number of cones, also in this species reach the visual centres of the brain.

This main conclusion would seem at variance with several studies with behavioural techniques in which the cat has been found to be colour blind (GUNTER 1954 b, MEYER, MILES and RATOOSH 1954). However, it is certain that the cat's retina possesses cones (*v. s.*, p. 9) which are regarded as responsible for the Purkinje shift of spectral sensitivity which can also be demonstrated in the feline ERG with high intensities (DODT and WALTHER 1958 b), as well as in single retinal elements (GRANIT 1947, BARLOW, FITZHUGH and KUFFLER 1957). Evidence from the investigations of GRANIT and his collaborators (GRANIT 1955 a), as well as from the results above shows that the cat is well equipped with blue sensitive receptors and perhaps also to some degree with green ones, while a red sensitive system is less prominent. GRANIT (1943) early pointed out that the cat's retinal structure showed certain similarities with the peripheral human retina (*cf.* WILLMER 1955) and that consequently, the cat might be regarded as protanope or, at least, highly protanomalous.

However, it is necessary to point out that all the spectral patterns demonstrated above would not necessarily be concerned with colour discrimination *per se*. This view, clearly stated by GRANIT (1947), is supported mainly by evidence from two sources: from observations in animals with pure rod retinae, and from sensitivity measurements in the rod-dominated peripheral retina of man.

Thus, the spectral sensitivity of the guinea pig, which has a pure rod retina, was investigated in 1942 by GRANIT. (As mentioned, SJÖSTRAND (1953) has demonstrated two kinds of rods in this animal.) The average retinal scotopic sensitivity showed a curve very similar to the rhodopsin distribution, as did the photopic except for a small hump in the blue. In light adaptation, several retinal elements demonstrated "modulator" curves with

single or coupled peaks at 450—470 mμ and at 530 mμ with a trough at 500 mμ in between. These curves bear a very close resemblance to the two low-intensity systems shown above in the cat. Apparently they do not subserve colour vision since MEYER, MILES and RATOOSH (1954) have demonstrated that guinea pigs are completely colour blind. DODT's (1956 a) findings in the dark adapted rabbit with the flicker method (*v. s.*, p. 88) should also be recalled here.

Receptor systems, which seem related to the characteristic variations of the low-intensity patterns found above in the cat, are, furthermore, found in the rod-rich human retinal periphery, in which colour discrimination is also very poorly developed. Thus, WEALE (1951, 1953 b) has shown that the sensitivity curve for this region, in contrast to that of the fovea, is doublehumped with peaks at 450—470 and at 530 mμ. This curve does, in fact, parallel the "visibility" curve of the light adapted cat (GUNTER 1954 b) rather strikingly.

There are obvious relations between the parallels drawn above and the many observations which indicate that the achromatic rod mechanism may be double (*v. s.*, p. 9—10). As mentioned, GRANIT in 1947 adduced evidence on the basis of adaptation data from the guinea pig and the cat that there are rods, and "cone-like" rods, which are responsible for a bend in the adaptation curves of these species. Findings in humans suffering from achromatopsia should also be recalled in this connection for such cases also demonstrate two phases in their adaptation curves (HECHT *et al.* 1938, LEWIS and MANDELBAUM 1943, SLOAN 1954, WALLS and HEATH 1954; *cf.* also YONEMURA and ISHISAKA 1955). Recently, RONCHI (1957) identified two interacting types of rod systems by means of ERG measurements in man. According to HECHT *et al.* (1938) there is reason to believe that the two mechanisms in achromatopsia have a spectral sensitivity closely similar to the rhodopsin absorption curve.

HECHT *et al.* (1948) and PIRENNE (1953) have suggested that the two rod systems, which have different thresholds, represent receptor mechanisms for gross and fine acuity in the scotopic state. PIRENNE (1953) suggested a pattern of large and small

retinal populations interacting at the ganglion cell level for these systems (*cf.* GRANIT and TANSLEY 1948). Following the line of evidence adduced above one would be justified in assuming a relationship between the two achromatic rod mechanisms in man and the two variations of spectral sensitivity described above for the cat at low intensities — which probably also exist in the rabbit and in the colour blind rod retina of the guinea pig.

It remains to be discussed, however, why the two achromatic systems show different spectral sensitivity patterns. If one assumes, with the authors quoted, that these systems subserve interacting large and small retinal receptor populations for gross and fine acuity, then an identical spectral sensitivity would not be expected. For the fine grain messages from the smaller populations would then always be swamped by those from the large ones. If, on the other hand, a comparatively smaller difference in spectral sensitivity existed between them, this would favor perception of acuity without necessarily implying wave-length discrimination. On the whole, the deviations from the rhodopsin curve of these systems were small (*cf.* logarithmic curves) as compared to the dramatic changes found in the photopic state, when there developed mainly a very large blue, a smaller green and only a suggestion of a peak in the orange-red. It seems a likely possibility that only peaks as large as the photopic blue and green would pertain to mechanisms subserving colour vision proper (*cf.* GUNTER 1954 a), and that these peaks with the suggested red one in the cat represent a rudimentary trichromatic wave-length discriminating sensory organ.

From the present investigation it is evident that mass responses from the visual centres of the brain, representing limited retinal receptor populations, are capable of differentiating scotopic and photopic mechanisms for light reception. Of necessity such responses must imply a considerable averaging of activity in a great number of single receptor conductor units. Records of such responses may therefore seem somewhat more representative than studies of single retinal elements in which usually only larger ganglion cells are selected (RUSHTON 1949, 1953 a).

In all probability it would be very rewarding to study retinal receptor functions in mass responses to flicker from the visual cortex in species with full colour vision (anthropoids and man) in which, furthermore, the spatial resolution of the retinal message in so much greater than in the cat.

Chapter V

General summary

1. In the present work a comparison has been made between spectral sensitivity as measured at the retina and at cerebral visual centres in the unanaesthetized (*cerveau isolé*) cat after initial dark adaptation and after additional adaptation to white light.

2. It has been confirmed that in the dark adapted state the retinal spectral sensitivity determined from the ERG mass responses to flicker demonstrates a 'scotopic blue-shift' (GRANIT and WIRTH 1953) when compared to the rhodopsin absorption curve.

3. In the dark adapted state, responses in the visual cortex to monochromatic flicker of different wave-lengths and of an intensity adapted to the retinal sensitivity curve, are grossly very similar although they demonstrate subtle differences in amplitude and latency. These differences suggest a heterogenous nature of scotopic light reception.

4. When On and Off responses in the visual cortex and other cerebral visual structures were studied as a function of intensity of monochromatic flicker, it was found that as the latency fell the amplitude increased with increasing intensity. Plots of latency and, especially, amplitude variations against intensity demonstrated, however, smaller differences between the wavelengths tested. The amplitude plots often showed shifts of gradients at certain intensities, *i. e.* the cortical responses would diminish above a certain intensity. Such shifts indicated that the cerebral visual responses resulted from interacting receptor-conductor mechanisms.

5. Spectral sensitivity distributions were calculated from energy reciprocals to successive steps of increasing equal ampli-

tude or falling latency in the wave-lengths tested. Spectral sensitivity was thus studied for steps of increasing light adaptation in flicker. From a great number of spectral curves certain patterns could be recognized in all experiments. In the initially dark adapted state, mainly two patterns were seen at lower intensities of flicker and a third pattern at higher intensities. The third pattern was brought out with especial clarity — reversibly though — by slight additional light adaptation.

6. The spectral patterns expressed by cerebral visual responses are unlikely to be caused by preretinal light absorption, tapetal reflection, light scatter, or systematic errors of the method. They must represent the spectral sensitivity of complex receptor systems in small retinal receptor populations.

7. Due to spatial resolution of the retinal messages these receptor systems can be analyzed with advantage from mass responses evoked in the visual centres of the brain. Accordingly, it was found that two points on the visual cortex, recorded from simultaneously, may express different spectral patterns on diffuse retinal illumination.

8. Records from all the main visual centres (striate area, superior colliculus and LGN) demonstrated that the main spectral patterns are projected to all these structures. It has thus been shown that sharply defined spectral messages, previously demonstrated in single ganglion cells of the cat's retina, may reach the visual centres to a full extent.

9. The two low-intensity patterns generally deviated less from the rhodopsin absorption curve. One contained a sharp spectral peak at 490 mμ which was often recorded in a relatively pure state, the other showed two peaks at 450—470 mμ and at 510—530 mμ with a trough at 490 mμ. The two low-intensity patterns probably represent two interacting receptor systems which coexist in the scotopic state and which may tentatively be ascribed to rods. In some experiments they were recorded differentially in On and Off components of the same response. The one with a peak at 490 mμ would seem to express increased absorption by rhodopsin relative to a blue-sensitive substance.

10. The third main pattern which deviated substantially from

the rhodopsin curve occurred at high intensities of flicker or with modest light adaptation. It has been ascribed to cones, since it contained a peak at 550 mμ. The maximum of this peak would correspond to that of the "photopic dominator" of GRANIT. Apart from this peak in the green, the high-intensity pattern contained a very high spectral peak in the blue (at 450—470 mμ, often also at 430 mμ) and a small hump in the orange (at 610 mμ).

11. The patterns found in cerebral visual structures show many similarities to previous observations on modulator functions in single retinal elements in the cat and other species. The high sensitivity at 470—490 mμ has been seen in electroretinographic work as a blue-shift in flicker. The ostensible lack of a single, rhodopsin-like, spectral sensitivity distribution in the scotopic state is probably explained by the introduction of some light adaptation by the flicker method.

12. The high-intensity pattern with its large blue and smaller green peak (and suggested hump in the orange) has been interpreted as a rudimentary trichromatic colour vision mechanism.

13. The two low-intensity patterns observed in the cat which interfere and lead to deviations from the rhodopsin absorption curve seem related to similar mechanisms in the guinea pig (which has two kinds of rods; SJÖSTRAND 1953) and the rabbit. They may also be related to the two rod systems present in man which have been shown to be achromatic in cases of achromatopsia. Such systems may be important for the perception of gross and fine acuity in the dark adapted state.

References

ABELSDORFF, G. Bemerkungen über das Auge der neugeborenen Katze, im Besonderen die retionale Sehzellenschicht. Arch. Augenh., *53*, 257—262, 1905.

ADAMS, R. G., LINSCHITZ, H. & KENNEDY, D. Kinetics of the photochemical conversion of rhodopsin in solution. Fed. Proc., *16*, 2, 1957.

ADRIAN, E. D. Visual responses in the cat and monkey. Proc. Physiol. Soc. 1941, 9 P, *in* J. Physiol., *100*, 1941.

— Rod and cone components in the electric responses of the eye. J. Physiol., *105*, 24—37, 1946.

ADRIAN, E. D. & MATTHEWS, R. The action of light on the eye. Part. III. The interaction of retinal neurones. J. Physiol., *65*, 273—298, 1928.

ALBE-FESSARD, D. Activités de projection et d'association du néocortex cérébral des mammifères. J. de physiol., *49*, 521—588, 1957.

APTER, J. T. Projection of the retina on superior colliculus of cats. J. Neurophysiol., *8*, 123—134, 1945.

BARLOW, H. B., FITZHUGH, R. & KUFFLER, S. W. Dark adaptation, absolute threshold and Purkinje shift in single units of the cat's retina. J. Physiol., *137*, 327—337, 1957.

BARRIS, R. W. Disposition of fibers of retinal origin in the lateral geniculate body. Course and termination of fibers of the optic system in the brain of the cat. Arch. Ophth., *14*, 61—70, 1935.

BARRIS, R. W., INGRAM, W. R. & RANSON, S. W. Optic connections of the diencephalon and midbrain of the cat. J. Comp. Neurol., *62*, 117—153, 1935.

BARTLEY, S. H. Temporal and spatial summation of extrinsic impulses with the intrinsic activity of the cortex. J. Cell. & Comp. Physiol., *8*, 41—62, 1936.

BISHOP, G. H. & CLARE, M. H. Sites of origin of electric potentials in striate cortex. J. Neurophysiol., *15*, 201—220, 1952.

— Organization and distribution of fibers in the optic tract of the cat. J. Comp. Neurol., *103*, 269—304, 1955.

BISHOP, G. H. & O'LEARY, J. L. Potential records from optic cortex of cat. J. Neurophysiol., *1*, 391—404, 1938.

— Electrical activity of the lateral geniculate of cats following optic nerve stimuli. J. Neurophysiol., *3*, 308—322, 1940.

BISHOP, G. H. & O'LEARY, J. L. The polarity of potentials recorded from the superior colliculus. J. Cell. & Comp. Physiol., *19*, 289—300, 1942 a.

— Factors determining the form of potential record in the vidinity of the synapses of the dorsal nucleus of the lateral geniculate body. J. Cell. & Comp. Physiol., *19*, 315—331, 1942 b.

BISHOP, P. O. Synaptic transmission. An analysis of the electrical activity of the lateral geniculate nucleus in the cat after optic nerve stimulation. Proc. Roy. Soc. London, ser. B, *141*, 362—392, 1953.

BISHOP, P. O. & EVANS, W. A. The refractory period of the sensory synapses of the lateral geniculate nucleus. J. Physiol., *134*, 538—557, 1956.

BISHOP, P. O., JEREMY, D. & LANGE, J. W. The optic nerve. Properties of a central tract. J. Physiol., *121*, 415—432, 1953 a.

BISHOP, P. O., JEREMY, D. & MCLEOD, J. G. Phenomenon of repetitive firing in lateral geniculate of cat. J. Neurophysiol., *16*, 437—447, 1953 b.

BISHOP, P. O. & MCLEOD, J. G. Nature of potentials associated with synaptic transmission in lateral geniculate of cat. J. Neurophysiol., *17*, 387—414, 1954.

BOLL, F. Zur Anatomie und Physiologie der Retina. Monatsber. Akad. Wiss. Berlin, 783—787, 1876.

BOYNTON, R. M. Stray light and the human electroretinogram. J. Opt. Soc. America, *43*, 442—449, 1953.

BREMER, F. Cerveau "isolé" et physiologie du sommeil. Compt. rend. Soc. de biol., *118*, 1235—1241, 1935.

BREMER, F. & STOUPEL, N. Analyse de la réponse élémentaire de l'aire visuelle du chat. J. de physiol., *48*, 410—411, 1956.

BRINDLEY, G. S. Human colour vision. Progress in Biophysics, *8*, 49—94, 1957.

BRODMANN, K. Vergleichende Lokalisationslehre der Grosshirnrinde, in ihren Prinzipien dargestellt auf Grund des Zellenbaues. Leipzig, 1909.

BRUESCH, S. R. & AREY, L. B. The number of myelinated and unmyelinated fibers in the optic nerve of vertebrates. J. Comp. Neurol., 77, 631—665, 1942.

BUCHER, V. M. & BÜRGI, S. M. Some observations on the fiber connections of the di- and mesencephalon in the cat. I. Fiber connections of the tectum opticum. J. Comp. Neurol., *93*, 139—172, 1950.

CAMPBELL, A. W. Histological studies on the localization of cerebral function. New York, 1905.

CHANG, H. T. Triple conducting pathway in the visual system and trichromatic vision. Année psychol., *50*, 135—144, 1951.

— Functional organization of central visual pathways. Proc. A. Res. Nerv. & Ment. Dis., *30*, 430—453, 1952.

— Fiber groups in the primary optic pathway of cat. J. Neurophysiol., *19*, 224—231, 1956.

CHANG, H. T. & KAADA, B. R. An analysis of primary response of visual

cortex to optic nerve stimulation in cats. J. Neurophysiol., *13*, 305—318, 1950.

CHIEVITZ, J. H. Untersuchungen über die Area centralis retinae Arch. f. Anat. u. Physiol. Anat. Abt., Suppl. 1889, 139—196.

CHOW, K. L. Failure to demonstrate changes in the visual system of monkeys kept in darkness or in colored lights. J. Comp. Neurol., *102*, 597—606, 1955.

CLAES, E. Contribution a l'étude physiologique de la fonction visuelle. I. Analyse oscillographique de l'activité spontanée et sensorielle de l'aire visuelle corticale chez le chat non anesthésié. Arch. int. de physiol., *48*, 181—237, 1939.

CLARE, M. H. & BISHOP, G. H. The intracortical excitability cycle following stimulation of the optic pathway of the cat. Electroencephalog. & Clin. Neurophysiol., *4*, 311—320, 1952.

— Properties of dendrites; apical dendrites of the cat cortex. Electroencephalog. & Clin. Neurophysiol., *7*, 85—98, 1955.

CLARK, W. E. L. The laminar pattern of the lateral geniculate nucleus considered in relation to colour vision. Docum. ophth., *3*, 57—64, 1949.

CLARK, W. E. L. & PENMAN, G. G. The projection of the retina in the lateral geniculate body. Proc. Roy. Soc. London, ser. B. *114*, 291—313, 1934.

COHN, R. A contribution to the study of color vision in cat. J. Neurophysiol., *19*, 416—423, 1956 a.

— Laminar electrical responses in lateral geniculate body of the cat. J. Neurophysiol., *19*, 317—324, 1956 b.

CREUTZFELDT, O. & AKIMOTO, H. Konvergenz und gegenseitige Beeinflussung von Impulsen aus der Retina und den unspezifischen Thalamuskernen an einzelnen Neuronen des optischen Cortex. Arch. f. Psychiat., *196*, 520—538, 1958.

DARTNALL, H. J. A. The interpretation of spectral sensitivity curves. Brit. M. Bull., *9*, 24—30, 1953.

— The visual pigments. London, 1957.

DAWSON, G. D. A summation technique for the detection of small evoked potentials. Electroencephalog. & Clin. Neurophysiol., *6*, 65—84, 1954.

DELL, P. Corrélations entre le système végétatif et le système de la vie de relation. Mesencéphale, diencéphale et cortex cérébral. J. de physiol., *44*, 471—557, 1952.

DEMOTT, D. W. & BOYNTON, R. M. Retinal distribution of entoptic stray light. J. Optic. Soc. America, *48*, 13, 1958 a.

— Sources of entoptic stray light. J. Optic. Soc. America, *48*, 120—125, 1958 b.

DENTON, E. J. & WYLLIE, J. H. Study of the photosensitive pigments in the pink and green rods of the frog. J. Physiol., *127*, 81—89, 1955.

DE VALOIS, R. L., SMITH, C. J., KITAI, S. T. & KAROLY, A. J. Response of

single cells in monkey lateral geniculate nucleus to monochromatic light. Science, *127*, 238—239, 1958.

DODT, E. Generalized inhibition of the retinal ganglion cells in relation to wave-length and state of adaptation. Acta Physiol. Scand., *36*, 219—228, 1956 a.

— Centrifugal impulses in rabbit's retina. J. Neurophysiol., *19*, 301—307, 1956 b.

— Geschwindigkeit der Nervenleitung innerhalb der Netzhaut. Experientia, *12*, 34—35, 1956 c.

DODT, E. & ELENIUS, V. Spektrale Sensitivität einzelner Elemente der Kaninchennetzhaut. Pflügers Arch., *262*, 301—306, 1956.

DODT, E. & ENROTH, C. Retinal flicker response in cat. Acta Physiol. Scand., *30*, 375—390, 1954.

DODT, E. and WALTHER, J. B. Netzhautsensitivität, Linsenabsorption und physikalische Lichtstreuung. Pflügers Arch., *266*, 167—174, 1958 a.

DODT, E. & WALTHER, J. B. Der photopische Dominator im Flimmer-ERG der Katze. Pflügers Arch., *266*, 175—186, 1958 b.

DONNER, K. O. The spike frequencies of mammalian retinal elements as a function of wave-length of light. Acta Physiol. Scand., *21*, suppl. 72, 1950.

— The spectral sensitivity of vertebrate retinal elements. Proc. N. P. L. Symposium on Visual Problems of Colour. Paper 20., Teddington, 1957. To be published.

DONNER, K. O. & GRANIT, R. Scotopic dominator and state of visual purple in the retina. Acta Physiol. Scand., *17*, 161—169, 1949.

ENROTH, C. The mechanism of flicker and fusion studied on single retinal elements in the dark-adapted eye of the cat. Acta Physiol. Scand., *27*, Suppl. 100, 1—67, 1952.

FRENCH, J. D. Some contributions of the physiological laboratory to problems in consciousness. I. congr. int. sci. neurol., Bruxelles, Seconde journée commune. Rapp. et disc., 97—109, 1957.

GLEES, P. The termination of optic fibres on the lateral geniculate body of the cat. J. Anat., *75*, 434—440, 1941.

GLEES, P. & CLARK, W. E. The termination of optic fibres in the lateral geniculate body of the monkey. J. Anat., *75*, 295—308, 1941.

GRANIT, R. Processes of adaptation in the vertebrate retina in the light of recent photochemical and electrophysiological research. Doc. Ophth., *1*, 7, 1938.

— Isolation of colour-sensitive elements in a mammalian retina. Acta Physiol. Scand., *2*, 93—109, 1941.

— Spectral properties of the visual receptor elements of the guinea pig. Acta Physiol. Scand., *3*, 318—328, 1941—1942.

— The spectral properties of the visual receptors of the cat. Acta Physiol. Scand., *5*, 219—229, 1943.

GRANIT, R. The colour receptors of the mammalian retina. J. Neurophysiol., *8*, 195—210, 1945.

— Sensory mechanisms of the retina. London, 1947.

— The mammalian colour modulators. J. Neurophysiol., *11*, 253—259, 1948.

— Effekter på retina vid stimulering i hjärnan. Nord. Med., *52*, 1674, 1954.

— Receptors and sensory perception. New Haven & London, 1955 a.

— Centrifugal and antidromic effects on ganglion cells of retina. J. Neurophysiol., *18*, 388—411, 1955 b.

GRANIT, R. & RIDELL, L. A. The electrical responses of light- and dark-adapted frogs' eyes to rhythmic and continuous stimuli. J. Physiol., *81*, 1—28, 1934.

GRANIT, R. & MUNSTERHJELM, A. The electrical responses of dark-adapted frogs' eyes to monochromatic stimuli. J. Physiol., *88*, 436—458, 1937.

GRANIT, R. & TANSLEY, K. Rods, cones and the localization of preexcitatory inhibition in the mammalian retina. J. Physiol., *107*, 54—66, 1948.

GRANIT, R. & WIRTH, A. A scotopic "blue shift" obtained by electrical measurements of flicker resonance. J. Physiol., *122*, 386—398, 1953.

GRÜSSER, O. J. Rezeptorpotentiale einzelner retinaler Zapfen der Katze. Die Naturwiss., *19*, 522, 1957.

GRÜSSER, O. J. & CREUTZFELDT, O. Eine neurophysiologische Grundlage des Brücke-Bartley-Effekts; Maxima der Impulsfrequenz retinaler und corticaler Neurone bei Flimmerlicht mittlerer Frequenzen. Pflügers Arch., *263*, 668—681, 1957.

GRÜSSER, O. J. & RABELO, C. Reaktionen retinaler Neurone nach Lichtblitzen. 1. Einzelblitze und Blitzreise wechselnder Frequenz. Pflügers Arch., *265*, 501—525, 1958 a.

GRÜSSER, O. J. & KAPP, H. Reaktionen retinaler Neurone nach Lichtblitzen II. Doppelblitze mit wechselnden Blitzintervall. Pflügers Arch., *266*, 111—129, 1958 b.

GUNTER, R. The spectral sensitivity of dark-adapted cats. J. Physiol., *118*, 395—404, 1952.

— The spectral sensitivity of light-adapted cats. J. Physiol., *123*, 409—415, 1954 a.

— The discrimination between lights of different wave lengths in the cat. J. Comp. & Physiol. Psychol., *47*, 169—172, 1954 b.

HAMDI, F. A. & WHITTERIDGE, D. The representation of the retina on the optic tectum of the pigeon. Quart. J. Exper. Physiol., *39*, 111—119, 1954.

HARTLINE, H. K. The neural mechanisms of vision. Harvey Lect., *37*, 39—68, 1941.

HECHT, S., SHLAER, S., SMITH, E. L., HAIG, C. & PESKIN, J. C. The visual

functions of a completely colorblind person. Am. J. Physiol., *123*, 94, 1938.

HECHT, S., SHLAER, S., SMITH, E. L., HAIG, C. & PESKIN, J. C. The visual functions of the complete colorblind. J. Gen. Physiol., *31*, 459—472, 1948.

HENSCHEN, S. E. Mémoire sur la base anatomique de la vision mono- et binoculaire. Remarques critiques à propos de la nouvelle hypothèse de R. Bárány. Trav. Lab. Rech. Biol. Madrid, *23*, 217, 1925.

— Lichtsinn- und Farbensinnzellen im Gehirn. Eine Studie. Klinische und anatomische Beiträge zur Pathologie des Gehirns. T. 8: 1, Stockholm 1930.

— Ueber spezifische Lichtsinn- und Farbensinnzellen im Gehirn. Acta Psychiat. & Neurol., *6*, 347—358, 1931.

HERNÀNDEZ-PEÓN, R., GUZMAN-FLORES, C., ALCARAZ, M. and FERNANDEZ-GUARDIOLA, A. Sensory transmission in visual pathway during "attention" in unanaesthetized cats. Acta Neurol. Latinoamer., *3*, 1—8, 1957.

HSIA, Y. & GRAHAM, C. H. Spectral sensitivity of the cones in the dark adapted human eye. Proc. Nat. Acad. Sc., *38*, 80—85, 1952.

HUBEL, D. H. Tungsten microelectrode for recording from single units. Science, *125*, 549, 1957.

— Single unit activity in visual cortex of the unanesthetized cat. Fed. Proc., *16*, 62, 1957.

HUNTER, J. & INGVAR, D. H. Pathways mediating metrazol induced irradiation of visual impulses. Experimental study in cat. Electroencephalog. & Clin. Neurophysiol., *7*, 39—60, 1955.

INGVAR, D. H. Colour differentiation in the visual cortex of the cat. Acta Psychiat. & Neurol. Scand., *31*, 163—166, 1956 a.

— Comparison of spectral sensitivity curves from the retina and central visual pathways in the cat. Abstr. of Comm. XX Intern. Physiol. Congr. Brussels, p. 459—460, 1956 b.

— Färgdiskrimination i kattens syncentra. Fysiologföreningen, Stockholm, dec. 1957.

— International Symposium on the Fundamental Mechanisms of the Chromatic Discrimination in Animals and Man. Paris July 25—29, 1958. Discussion. To be Published.

INGVAR, D. H. & HUNTER, J. Influence of visual cortex on light impulses in the brain stem of the unanaesthetized cat. Acta Physiol. Scand., *33*, 194—218, 1955.

IVES, H. E. Studies in the photometry of lights of different colours I, II, IV and V. Philos. Mag., *24*, pp 149, 352, 744, 843, 853, 1912.

JOHNSON, E. P. & CORNSWEET, T. N. Electroretinal photopic sensitivity curves. Nature, *174*, 614—615, 1954.

JUNG, R., BAUMGARTEN, R. v. & BAUMGARTNER, G. Mikroableitungen von einzelnen Nervenzellen im optischen Cortex der Katze: Die litchtaktivierten B-Neurone. Arch. Psychiat., *189*, 521—539, 1952.

KOLMER, W. Die Netzhaut. Handb. d. Mikr. Anat. d. Mensch., Ed. W. v. Möllendorff *3*: 2, 295—467, 1936.

KUFFLER, S. W. Discharge patterns and functional organization of mammalian retina. J. Neurophysiol., *16*, 37—68, 1953.

KÜHNE, W. Chemische Vorgänge in der Netzhaut. *in* Handbuch v. Physiol., Prog. von L. Hermann, *3*, 235—242, 1879.

KÖNIG, A. Über den menschlichen Sehpurpur und seine Bedeutung für das Sehen. Sitzungsb. Akad. Wiss., Berlin, 577—598, 1894.

LENNOX, M. A. Geniculate and cortical responses to colored light flash in cat. J. Neurophysiol., *19*, 271—279, 1956.

— Single fiber responses to electrical stimulation in cat's optic tract. J. Neurophysiol., *21*, 62—69, 1958 a.

— The On responses to colored flash in single optic tract fibers of cat Correlation with conduction velocity. J. Neurophysiol., *21*, 70—84, 1958 b.

LENNOX, M. A. & MADSEN, A. Cortical and retinal responses to colored light flash in anesthetized cat. J. Neurophysiol., *18*, 412—424, 1955.

LEWIS, S. D. & MANDELBAUM, J. Achromatopsia: Report of three cases. Arch. Ophth., *30*, 225—231, 1943.

LUDVIGH, E. & MCCARTHY, E. F. Absorption of visible light by refractive media of human eye. Arch. Ophth., *20*, 37—51, 1938.

MACNICHOL, E. F., MACPHERSON, L. & SVAETICHIN, G. Studies on spectral response curves from the fish retina. N. P. L. Symposium on Visual Problems of Colour, Paper 39., Teddington 1957. To be published.

MADSEN, A. & LENNOX, M. A. Response to colored light flash from different areas of optic cortex and from retina in anesthetized cat. J. Neurophysiol., *18*, 574—582, 1955.

MAGOUN, H. W. An ascending reticular activating system in the brain stem. Arch. Neurol. & Psychiat., *67*, 145—154, 1952.

MAGOUN, H. W. & RANSON, S. W. The central path of the light reflex; study of effect of lesions. Arch. Ophth., *13*, 791—811, 1935.

MALIS, L. I. & KRUGER, L. Multiple responses and excitability of cat's visual cortex. J. Neurophysiol., *19*, 172—186, 1956.

MARQUIS, D. G. Effects of removal of the visual cortex in mammals, with observations on the retention of light discrimination in dogs. A Research Nerv. & Ment. Dis., Proc. *13*, 558—592, (1932) 1934.

MARSHALL, W. H. & TALBOT, S. A. 1941, quoted from GLEES, P. Morphologie und Physiologie des Nervensystems, Stuttgart, 1957.

MARSHALL, W. H., TALBOT, S. A. & ADES, H. W. Cortical response of the anesthetized cat to gross photic and electrical afferent stimulation. J. Neurophysiol., *6*, 1—15, 1943.

MEYER, D. R., MILES, R. C. & RATOOSH, P. Absence of color vision in cat. J. Neurophysiol., *17*, 289—294, 1954.

MILES, R. C., RATOOSH, P. & MEYER, D. R. Absence of color vision in guinea pig. J. Neurophysiol., *19*, 254—258, 1956.

MINKOWSKI, M. Experimentelle Untersuchungen über die Beziehungen der Grosshirnrinde unter der Netzhaut zu den primären optischen Zentren, besonders zum Corpus geniculatum externum. Arb. hirnanat. Inst., Zürich, 7, 255—362, 1913.

— Über den Verlauf, die Endingungen und die zentrale Representation von gekreuzten und ungekreuzten Sehnervenfasern bei einigen Säugetieren und bei Menschen. Schw. Arch. Neurol. & Psychiat., *6*, 201—252, 1920.

MOTOKAWA, K., IWAMA, K. & EBE, M. Retinal color processes in cats. Jap. J. Physiol., *2*, 198—207, 1952.

MOTOKAWA, K., TUKAHARA, S. & EBE, M. Cortical reponse to intermittent stimulation with colored light in the cat. Jap. J. Physiol., *3*, 191—199, 1953.

MOTOKAWA, K., OIKAWA, T. & TASAKI, K. Receptor potential of vertebrate retina. J. Neurophysiol., *20*, 186—199, 1957.

O'LEARY, J. L. A structural analysis of the lateral geniculate nucleus of the cat. J. Comp. Neurol., *73*, 405—430, 1940.

— Structure of the area striata of the cat. J. Comp. Neurol., *75*, 131—164, 1941.

O'LEARY, J. L. & BISHOP, G. H. Electrical activity of the lateral geniculate nucleus of the cat. Proc. Am. Physiol. Soc. *in* Am. J. Physiol., *126*, 594—595, 1939.

PIRENNE, M. H. The absolute sensitivity of the eye and the variation of visual acuity with intensity. Brit. M. Bull., *9*, 61—67, 1953.

POLJAK, S. An experimental study of the association callosal and projection fibers of the cerebral cortex of the cat. J. Comp. Neurol., *44*, 197—258, 1927.

— Projection of the retina upon the cerebral cortex, based upon experiments with monkeys. A. Research Nerv & Ment. Dis., Proc. *13*, 535—557, 1932.

POLYAK, S. L. The retina. Chicago, 1941.

— The vertebrate visual system. Chicago, 1957.

RAMÓN y CAJAL, S. Studien über die Sehrinde der Katze. J. f. Psychol. u. Neurol., *29*, 161—181, 1923.

— Histologie du système nerveux de l'homme et des vertébrés. T. 1—2. Reimpr. Madrid, 1952—1955.

ROGGENBAU, C. & WETTHAUER, A. Über die Durchlässigkeit der brechenden Augenmedien für langwelliges Licht nach Untersuchung am Rindesauge. Klin. Monatsbl. f. Augenh., *79*, 456—464, 1927.

RONCHI, L. On the electrical response of the darkadapted human retina to stimuli presenting different time-luminance distributions. Atti Dell Fondazione Giorgico Ronchí, *12*, 560—568, 1957.

RUSHTON, W. A. H. The structure responsible for action potential spikes in the cat's retina. Nature, *164*, 743—744, 1949.

RUSHTON, W. A. H. Electric records from the vertebrate optic nerve. Brit. M. Bull., *9*, 68—74, 1953 a.

— The measurement of rhodopsin in the living eye. Acta Physiol. Scand., *29*, 16—18, 1953 b.

— Human cone pigments. Proc. N. P. L. Symposium on Visual Problems of Colour. Paper 1., Teddington 1957. To be published.

SCHULTZE, M. Zur Anatomie und Physiologie der Retina. Arch. Mikr. Anat., *2*, 175—286, 1866.

SJÖSTRAND, F. S. The ultrastructure of the inner segments of the retinal rods of the guinea pig eye as revealed by electron microscopy. J. Cell. & Comp. Physiol., *42*, 45—70, 1953.

SLOAN, L. L. The effect of intensity of light, state of adaptation of the eye, and size of photometric field on the visibility curve. Psychol. Monog., *38*, 1—87, 1928.

— Congenital achromatopsia: A report of 19 cases. J. Optic. Soc. America, *44*, 117—128, 1954.

SOLNITZKY, O. & HARMAN, P. J. A comparative study of the central and peripheral sectors of the visual cortex in Primates, with observations on the lateral geniculate body. J. Comp. Neurol., *85*, 313—419, 1946.

STILES, W. S. The directional sensitivity of the retina and the spectral sensitivities of the rods and cones. Proc. Roy. Soc. London, Ser. B. *127*, 64—105, 1939.

ST. GEORGE, R. C. C. The interplay of light and heat in bleaching rhodopsin. J. Gen. Physiol., *35*, 495—517, 1952.

SVAETICHIN, G. Spectral response curves from single cones. Acta Physiol. Scand., *39*, Suppl. 134, 19—46, 1956 a.

— Receptor mechanisms for flicker and fusion. Acta Physiol. Scand., *39*, Suppl. 134, 49—54, 1956 b.

— The cone function related to the activity of retinal neurons. Acta Physiol. Scand., *39*, Suppl. 134, 69—91, 1956 c.

TALBOT, S. A. & MARSHALL, W. H. Binocular interaction and excitability cycles in cat and monkey. Proc. Am. Physiol. Soc. *in* Am. J. Physiol., *133*, 467—468 P, 1941.

TELLO, F. Disposición macroscópica y estructura del cuerpo geniculado externo. Trab. Lab. Invest. Biol. Madrid, *T. 3*, 39—62, 1904.

THUMA, B. D. Studies on the diencephalon of the cat. I. The cyto-architecture of the corpus geniculatum laterale. J. Comp. Neurol., *46*, 173—200, 1928.

WALD, G., BROWN, P. K. & SMITH, P. H. Iodopsin. J. Gen. Physiol., *38*, 623—681, 1955.

WALLS, G. L. The vertebrate eye and its adaptive behaviour. Cranbrook Inst. of Sc. Bulletin, *19*, 1932.

— The lateral geniculate nucleus and visual histophysiology. Univ. of Calif. Publ. in Psychol., *9*, 1—100, 1953.

WALLS, G. L. & HEATH, G. G. Typical total color blindness reinterpreted. Acta Ophth., *32*, 253—297, 1954.

WEALE, R. A. The foveal and para-central spectral sensitivities in man. J. Physiol., *114*, 435—446, 1951.

— The spectral reflectivity of the cat's tapetum measured *in situ*. J. Physiol., *119*, 30—42, 1953 a.

— Spectral sensitivity and wave-length discrimination of the peripheral retina. J. Physiol., *119*, 170—190, 1953 b.

— Colour vision in the peripheral retina. Brit. M. Bull., *9*, 55—60, 1953 c.

— Photochemical reactions in the living cat's retina. J. Physiol., *122*, 322—331, 1953 d.

— Slow and rapid regeneration in the living cat's retina. J. Physiol., *122*, 11 P, 1953 e.

— Light absorption in the crystalline lens of the cat. Nature, *173*, 1049—1050, 1954.

— Retinal summation and human visual thresholds. Nature, *181*, 154—156, 1958.

WILLMER, E. N. A physiological basis for human colour vision in the central fovea. Docum. ophth. *9*, 235—313, 1955.

WIRTH, A. Electroretinographic evaluation of the scotopic visibility function in cats and albino rabbits. Acta Physiol. Scand., *29*, 22—30, 1953.

VRIES, H. L. DE. The animosity curve of the eye as determined by measurements with flicker-photometer. Physica, *14*, 319—348, 1948.

WRIGHT, W. D. Researches on normal and defective colour vision. London, 1946.

WULFF, V. J., ABRAHAMSON, W. E., LINSCHITZ, H. & ADAMS, R. G. Light-induced transient changes in the absorption spectrum of rhodopsin in solution. Fed. Proc., *16*, 139, 1957.

YONEMURA, D. & ISHISAKA, N. On the electrical excitability of the eye in congenital achromatopsia (with Motokawa's method). Tokohu J. Exp. M., *62*, 377—389, 1955.

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